ASSESSMENT OF HUMAN HEALTH RISK OF REPORTED SOIL LEVELS OF METALS AND RADIONUCLIDES IN PORT HOPE

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Background

This scientific document represents an assessment of potential exposure and environmental health risks associated with elevated levels of eleven metals and three radionuclides in soil. The substances assessed are listed in the table below. The specific site in question is the area surrounding the Eldorado Resources Ltd. (Cameco) facility in the town of Port Hope, Ontario. The assessment examines measured and computer modelled levels of soil contamination. These levels in soil stem from an extensive soil sampling survey conducted by the Ministry of the Environment in 1986 and 1987 which is reported in the MOE document "Phytotoxicology Assessment surveys in the Vicinity of Eldorado Resources Ltd., Port Hope, 1986 and 1987."

Metals and Radionuclides Assessed

METALS	RADIONUCLIDES
Arsenic	Ra(226)
Antimony	Pb(210)
Uranium	U(238)
Lead	
Chromium	
Copper	
Nickel	
Cadmium	
Cobalt	
Selenium	
Zinc	

The assessment was developed because the reported levels of a number of metals exceeded the Ministry of the Environment's Upper Limit of Normal guidelines and Guidelines for Decommissioning of Industrial Sites. In addition, the survey identified three radionuclides (^{226}Ra, ^{210}Pb and ^{238}U) as consistently exceeding reported background levels. The undertaking of the analysis is in keeping with the concept of application of upper limit of normal guidelines, which if exceeded prompt further investigation on a case-by-case basis.

Multimedia Risk Assessment Approach

It is well established that humans can be exposed to metals from a

variety of sources including drinking water, food, ambient air, soils/dusts, and consumer products. In order to evaluate the health significance of exposure via a particular pathway or source (in this case, soil) it is necessary to begin to understand the total exposure picture from all routes. The multimedia risk assessment approach, which considers total exposure through multiple pathways, was utilized to evaluate human exposure to each of the metals.

Exposure estimates for each significant pathway are developed for adults and young children. Young children are assessed as a special population subgroup because of their greater contact with and direct ingestion of soil and dusts. As well, children may be more sensitive to the effects of certain contaminants. The relative contribution of soil/dust ingestion to total exposure is assessed relative to the intake from other pathways (food, air, drinking water). The latter are modelled utilizing existing monitoring information or assumed concentrations together with values for human receptor characteristics (e.g daily breathing volumes). Average exposures for each pathway are integrated to provide an estimate of potential total exposure.

The approach utilizes the available information on contaminant levels in various media. Actual levels in Port Hope for media other than soil, may differ from what is assumed/predicted. This information is then utilized to predict exposure for the typical individual in either the child or adult age groups. It should be recognized that individual exposures patterns will vary widely depending on many factors such as diet, age, occupation, sex and personal behaviour choices. The exposure estimates provided must be viewed in light of these provisions.

With respect to discrete exposures from the soils in question, human exposure and intake may occur through multiple pathways from the soil source. The most obvious for metals in soil are incidental soil/dust ingestion for children at play and adults engaging in certain activities (e.g landscaping), dermal contact and indirect intake through the consumption of homegrown vegetables.

Intake via soil ingestion is quantitatively assessed for each metal. The primary exposure scenario is one based upon an assumption of continual daily exposure to the reported levels. This does not take into account indoor/outdoor exposure, seasonal variability, residence time in areas or land use. As such, calculated intakes will tend to overestimate actual exposures. Consideration of exposure under other scenarios is considered for specific metals in soils from recreational areas.

A question which is generally associated with soil contamination is that of the possible additional exposures and concomitant risk to people who grow and consume vegetables on residential sites. The survey did not specifically sample levels in homegrown vegetables and therefore a determination of risk is quite difficult. However,

as an attempt to look at this exposure scenario, a limited analysis was undertaken. Estimates of exposure through consumption of backyard vegetables are determined through the modelling of levels of these substances in vegetables. Because such methods are of limited reliability these estimates are not included in the total estimates of daily intake and do not appear in the exposure assessment section for individual metals. Rather they are provided (see Appendix II) to allow for comparison to other exposure pathways and the additional fraction of exposure (i.e. % of average daily intake) to that metal that homegrown produce consumption might roughly represent.

Qualitative consideration of the dermal pathway is provided for the metals. This generally is only a minor pathway for inorganics and would represent only a trivial contribution to total exposure.

To characterize risk, the calculated intakes for adults and children are compared against existing acceptable intakes or oral reference doses for each metal. This applies to those toxic effects which exhibit a threshold. For carcinogenic metals, where no "safe" level or threshold has been established, associated risk levels are determined.

For the radionuclides, exposure is assesed through the most significant environmental routes of intake from soil (ingestion of soil and homegrown vegetables).

RESULTS AND DISCUSSION

Arsenic

Arsenic is a known human carcinogen. Ingestion of inorganic arsenic is associated with skin cancer in humans. The determination of risk related to soil exposure is based upon this effect.

In Ontario, including Port Hope, average intakes of arsenic are expected to occur predominantly through the food pathway, accounting for approximately 90% of daily exposure. The average measured arsenic level in soil in the Port Hope surveys is 20 µg/g, with measured levels ranging from 2 to 234 µg/g. The majority of measurements fall below the arithmetic mean value as the data tend to be log-normally distributed. The estimated average intake from soil based on the mean arsenic level would increase the total absorbed intake above background by 0.8 - 9.5 % for children and 0.2 -2% for adults. The predicted relative contribution of soil/dust to total daily intake for children and adults is 9% and 2% respectively.

Risk from these arsenic exposures is characterized in two manners: 1.) treating arsenic as a carcinogen without threshold and calculating risk levels associated with potential exposures and 2.) treating arsenic as a carcinogen with a threshold for these effects

and comparing modelled soil exposure to predicted toxicity thresholds. Two approaches are taken because the body of world expert opinion differs on the question of arsenic carcinogenicity by ingestion.

For considering arsenic as a carcinogen without threshold, calculations are based upon U.S. EPA skin cancer potency factor for ingested inorganic arsenic. The average daily intakes from soil/dust yield predictions of incremental frequency (above background) of skin cancer of 0.0006/year (0.05 over lifetime) for a group or population the size of Port Hope (approximately 10,000). This assumes daily exposure to these concentrations over a lifetime. Calculated average individual incremental risks from arsenic in soil/dust are estimated at 1×10^{-5}. The corresponding risk level at 50 µg/g (highest SYMAP[1] predicted isopleth) is 2.5×10^{-5}. Concentrations of greater than 50 µg/g are confined to two relativley small areas of Port Hope according to the SYMAP contours. A level of 110 µg/g (measured on a residential property) would have a corresponding risk level of approximately 5×10^{-5}. These risk levels are within the order of magnitude of risk generally thought of as negligible. Further, the potency factor utilized is derived from modelling methods which are generally recognized as conservative and therefore may tend to overestimate risk. In comparison, using the same potency factor, the risk level associated with daily ingestion of food (not homegrown) is 2×10^{-4}, which is more than 10 times greater than the average soil/dust risk.

With respect to the two small areas designated as " >50 µg/g" on the SYMAP projections, it is difficult to estimate risk without more definitive information on actual soil levels. If residential soil levels were above this value in these areas, risk levels may be correspondingly higher.

If risk is assessed considering arsenic as a carcinogen which exhibits threshold, comparing total arsenic intake (child and adult) from all pathways to threshold levels indicate arsenic intakes are within the range of detoxification suggested by a model of arsenic metabolism. Estimated exposures are also below the provisional maximum permissable intake levels established by the World Health Organization.

In summary, arsenic concentrations in soil were examined utilizing two approaches. Estimated intakes from contact with these soils yield risk estimates in the range generally considered negligible and below the WHO permissable intake.

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1 SYMAP is an acronym for synigraphic computer mapping. Used in conjunction with monitoring data it can produce contamination contour maps of a site location

Antimony

A complete quantitative assessment of antimony exposures was not possible given the lack of dietary exposure data. Confidence in the only available reference dose is also low. Precise conclusions based on quantitative methods cannot be reached. Qualitatively, it may be considered that antimony is thought to behave biologically and chemically in a similar manner to arsenic but to be substantially less toxic. It is also likely that normal dietary intake would account for the bulk of exposure. The maximum concentration was associated with an area adjacent to the west bank of the Ganaraska River. Taking into consideration recreational exposure times, soil/dust intakes were calculated and are below the reference dose value. Based on these factors, antimony concentrations are not anticipated to pose a hazard in these areas.

Uranium

Exposure to uranium can result in two basic categories of effects: those associated with the radiobiological properties of uranium and those associated with its chemical properties. The available toxicological information suggests in general that chemical and not radiological effects may be assumed to be the limiting factor with respect to human health at environmental levels of exposure. The sensitive health endpoint is effects on the kidney.

The principle health effect considered in the development of reference doses for oral exposure to uranium in humans is nephrotoxicity or kidney effect. Comparison of both discrete soil intake estimates and total daily intakes within current acceptable daily intakes/reference doses indicate that these exposures are well below the most conservative limits. Based on nephrotoxicological effects, it is concluded that exposure to uranium in soil would not be predicted to pose an untoward health hazard in these areas.

Lead

Young children are generally thought to be the most sensitive receptors of lead and will have the greatest potential contact with soils. The principle potential health effects associated with lead at low levels of exposure involve changes in heme synthesis and subtle neurobehavioural deficits. There may not be a threshold or safe level for neurobehavioural/developmental effects in children. In other words any exposure to lead may carry some degree of risk. People are exposed to lead through various pathways (food, water, consumer products, etc) everyday.

The average concentration of lead in soil in Port Hope is 140 µg/g, including all sites. Measured levels are typically less than 200 µg/g, with most concentrations less than 100 µg/g. Estimated exposures for children to lead in soil in Port Hope would be

predicted on average to be less than similar exposures for other urban Ontario locations, where lead levels of several hundred µg/g are not unusual. Average soil/dust exposures may account for 20-40% of daily exposure of children, with 50-75% through food. The total combined exposures from all media will not be greater than the WHO provisional tolerable daily intake, although the margin of safety cannot be considered large.

According to the SYMAP projections, concentrations of greater than 500 µg/g are limited to two small confined areas. Modelled intake from soil at residential sites at 500 µg/g would be about 75% of the tolerable intake (this assumes continuous daily exposure to that area). Taken together with other exposures, total intakes would be estimated at approximately 1.4 times the suggested intake limit.

As with antimony, the highest concentrations of lead are found in the area of the west bank of the Ganaraska River. Intakes from soil ingestion alone, based on the more reliable measurement (1300 µg/g) and adjusted for time spent in the area, would fall within intake limits. However, if soil concentrations are several thousand µg/g (as one highly variable sample taken in 1986 possibly suggests), then exposures could exceed tolerable intakes by several fold. Furthermore, with respect to the remediation of contamination around existing facilities, the Royal Society of Canada has recommended that levels of up to 1000 µg/g should be acceptable for parklands and other areas to which children may have intermittent access.

In summary, given the limited degree of contamination, the soil levels of lead in these areas in general are not anticipated to pose a hazard. Extended contact with the site on the riverbank may result in exposures above tolerable intakes depending on the extent and level of contamination.

Chromium, Copper, Cadmium, Nickel, Cobalt and Selenium

The estimated average levels of chromium, copper, cadmium, nickel, cobalt and selenium in soil are not predicted to pose any appreciable health risk to persons living in these areas. This is based on:

1.) average estimated exposure via soil would result in intakes below the human oral acceptable intakes and reference doses for these metals.

2.) soil/dust intakes represent less than 1% of the average exposure contribution.

3.) nickel, chromium, copper and cobalt are essential dietary nutrients.

Zinc and Iron

Iron is not evaluated in the quantitative risk assessment because of the low hazard associated with iron intake and its known nutritional benefits.

Zinc is qualitatively assessed given its low level of toxicity to humans. Based on a comparison to recommended nutrient intakes for Canadians, the reported zinc levels are not believed to present any health implications.

Vegetable Consumption

In the absence of measured levels of metals in backyard fruits and vegetables, modelled intakes of the metals through homegrown produce are presented. These values are very crude estimates and have not been included in the estimates of total daily intakes. Consumption of backyard vegetables grown in contaminated soil will increase the intake levels of individuals. The suggested intakes are compared to the estimated total daily exposures from other routes. The additional exposures range from less than 1% for chromium to roughly 25% more for uranium in adults (see appendix II).

RADIONUCLIDES ASSESSMENT

If ingested, Ra(226) and Pb(210) are stored in bone and present the potential risk of induction of bone cancer. The major ingestion pathways from soil are the ingestion of contaminated soil and the consumption of garden produce. It is concluded under reasonable assumptions about the quantities of soil and produce ingested, the annual radiation dose attributable to these radionuclides will be less than several percent of the population limits.

With respect to U(238), chemical toxicity is thought to be the limiting effect for environmental exposures, although radiobiological effects can occur. The estimated average soil intake for a child is less than 0.1% of population exposure limits and occupational annual limits of intake.

CONCLUSIONS

Risk assessment methods are applied to the question of health implications of contaminated soil in the Port Hope area. Soil-related as well as other pathways of exposure are considerd.

Exposures to the reported levels of uranium, antimony, chromium, copper, nickel, cadmium, cobalt, selenium, and zinc in Port Hope soils are not expected to result in adverse health consequences.

Oral exposure to arsenic in soil at the reported levels is estimated to result in incremental cancer risk levels in the negligible range (10^{-5}). Estimated exposures also fall well below suggested toxic thresholds for arsenic. For the two small areas areas within the >50 µg/g isopleth, assessment of exposure is difficult without more definitive data on soil concentrations in these zones.

Contamination of soils with lead is overall quite limited. In general, the reported soil levels of lead are not anticipated to pose a hazard. The site with the highest concentrations of lead is located on the west bank of the Ganaraska River, a popular fishing area. Depending on the level and extent of contamination, as well as degree of contact with the site, potential exposures could exceed tolerable intakes for children.

Exposures to the radionuclides Ra(226), Pb(210) and U(238) in soil at the reported levels are estimated to fall well within recommended population limits.

PREFACE

This document represents the work of the Risk Assessment Unit, Hazardous Contaminants Branch (HCB) of the Ontario Ministry of the Environment and the Health Studies Service of the Ontario Ministry of Labour. The document consists of two major components. The detailed multimedia risk assessment of inorganics was prepared by toxicological risk assessment staff of HCB. The component dealing with radiological risk was prepared by MOL medical staff with expertise in the health physics area. Valuable contributions to the inorganics assessment were also provided by MOL staff.

It is important to understand that there are a number of uncertainties associated with the process of risk assessment arising from the lack of empirical scientific data in numerous areas. The risk assessment approach presented utilizes assumptions which are generally considered conservative predictive methods. Actual risks may in fact be much lower than those described, although this cannot be precisely determined. For those reasons, risk estimates should be considered not as true/actual risks but as estimates to be utilized as a crude measure of potential impact. The risk assessment, herein, is specific to the sites in question and should not be directly applied to interpretation of other situations or scenarios.

INTRODUCTION

In 1986 and 1987, the Phytotoxicology Section of the Ontario Ministry of the Environment conducted soil and vegetation surveys in the vicinity of Eldorado Nuclear Ltd. in the city of Port Hope. These surveys were part of annual assessments carried out in this area. The results of these surveys were summarized in a separate draft report (MOE, 1989b). It was determined that surface soil concentration for a number of inorganic substances (uranium, antimony, copper, nickel, lead, arsenic, zinc, chromium and cobalt) exceeded the Phytotoxicology Upper Limit of Normal Guidelines, and that the substances that exceeded the guidelines most often and by the greatest amount were uranium, arsenic and antimony. Cadmium and selenium levels were are also reported in soil. Furthermore, a number of radionuclides were analyzed in soil, and ^{226}radium (^{226}Ra), ^{210}lead (^{210}Pb) and ^{238}uranium (^{238}U) were found at activities which exceed the reported background levels. The soil survey report indicted that the implications for human health from exposure to soil contaminated with these elements and radionuclides at the concentrations measured had not previously been fully examined.

In light of these findings it was recommended that the soil survey data should be reviewed by the appropriate environmental health experts prior to release of the report. This is in keeping with the concept of application of upper limit of normal (ULN) guidelines. These guidelines do not represent maximum desirable or allowable levels of contaminants, but rather serve as levels which, if exceeded, prompt further investigation on a case-by-case basis to determine the significance, if any, of the above- normal concentrations. This current assessment of risk represents such a further investigation. It is meant to examine in some detail, utilizing current toxicological information and multi-pathway modelling of human exposure, the possible human health implications of the metal and radionuclides in the soil at these sites. The quantitative risk assessment methods described below were used to consider the potential intakes of contaminants from soil/dust and to assess them against other common exposure pathways (food, air, drinking water). These findings will contribute to defining the need for future action.

This particular document is divided into two components. The first of these deals with assessment of chemical risk from inorganics with consideration of multimedia exposures to these metals. The second component is concerned with the assessment of the radionuclides in question and focuses on the direct soil ingestion and vegetable consumption pathways, as these are the major pathways for consideration in assessing the possible radiobiological risks.

Part I HEALTH RISK ASSESSMENT OF INORGANICS

Overview of Risk Assessment

In assessing environmental health risks, whether in a site-specific or general population context, it is increasingly recognized that an integrated assessment of exposure through multiple pathways is necessary to understand the total risk to receptors as well as the contributing risk posed by the media under examination. The general methodology utilized is based on conventional models of risk assessment (see Figure 1). The risk assessment for each metal consists of four principle components. These are: (1) hazard identification (toxicological profile), (2) dose-response assessment, (3) human exposure assessment and (4) risk characterization. This framework is aimed at determining what the magnitude of exposures may be through various pathways for different subgroups and whether or not adverse or undesirable effects from such chemicals would be expected from such exposures.

1. Hazard Identification

Hazard identification is the stage of risk assessment in which there is preliminary identification of the potential adverse health effects from a chemical in order to allow preliminary judgement as to the level of concern for the contaminant.

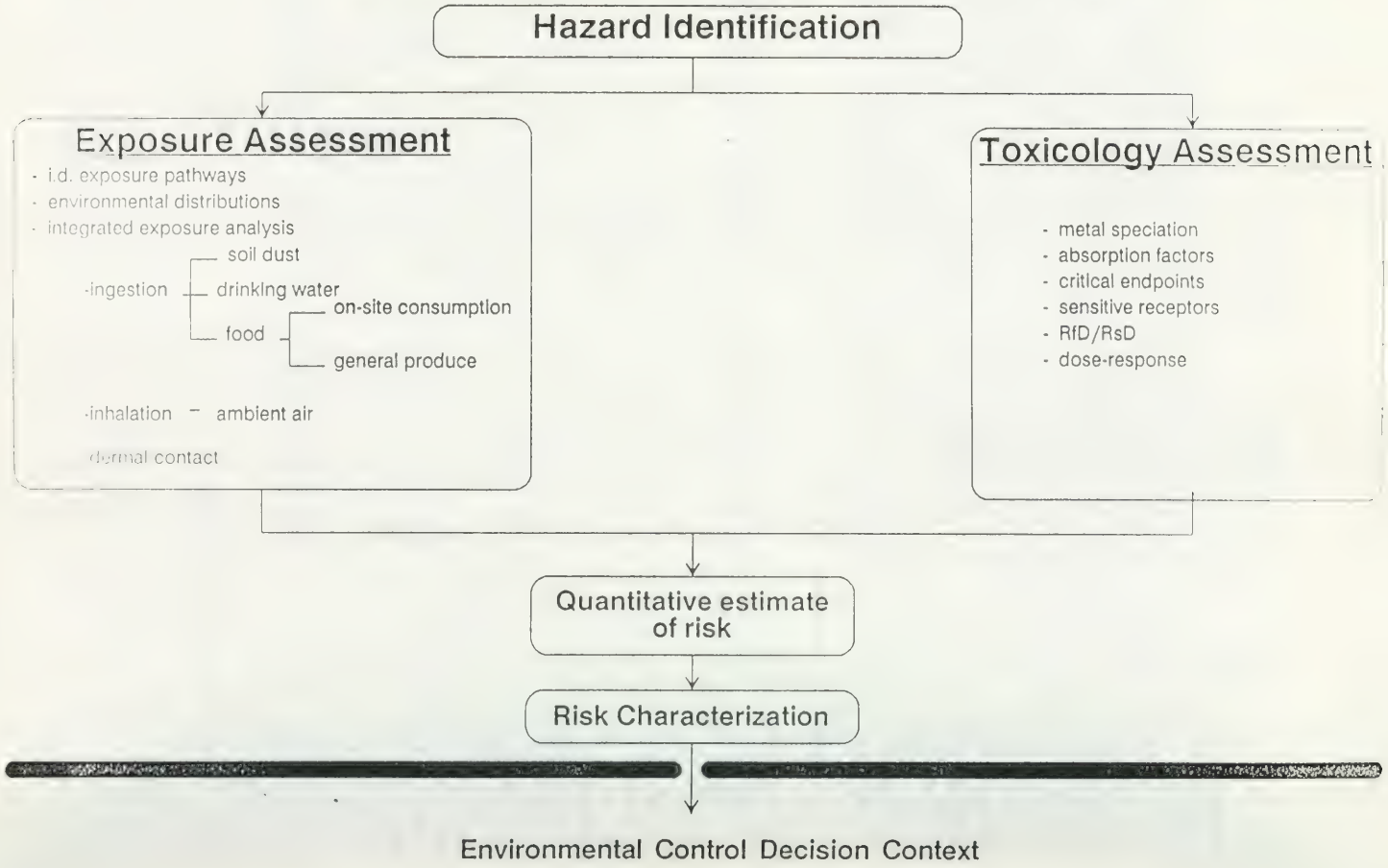
This process identifies qualitatively the type of adverse health effect (e.g. cancer, neurotoxicity) associated with this substance in the scientific literature. This can include animal toxicology studies, epidemiological studies, and where data are lacking, a comparison of the chemical structure/activity to that of substances already known. The information provided here is not meant to represent a comprehensive review of all the available information, but rather a synopsis of the more current and relevant summary information. Information sources from which toxicity data were obtained include computerized databases such as the Registry of Toxic Effects of Chemical Substances (RTECS, 1987), TOXLINE, Hazardous Substances Database (HSDB), United States Environmental Protection Agency (U.S. EPA) Integrated Risk Information (IRIS, 1990), Chemical Evaluation Search and Retrieval System (CESARS) and the Chemical Abstract Service (CAS). Other information sources included review and regulatory documents from the ministry and other agencies and where indicated, the original scientific literature.

2. Dose-Response Assessment

Dose-response assessment is the determination of the relationship between the magnitude of exposure routes and the probability of the occurrence of effects on human and non-human biota. For non-carcinogenic effects, dose-response information is generally utilized by regulatory agencies to develop acceptable exposure

FIGURE 1:

RISK ASSESSMENT FRAMEWORK



levels (e.g. acceptable daily intakes (ADI) and reference doses (RfD)) below which adverse effects are not expected to occur.

In quantitative risk assessment for cancer, carcinogenicity is often expressed in terms of a potency slope, which is then used to calculate the probability (risk) of cancer associated with a given exposure level.

In this assessment, there is no attempt to develop such relationships from reported studies; rather this information is adopted from credible regulatory bodies.

For both carcinogenic and non-carcinogenic endpoints, potency factors and reference dose values are gathered from various agencies, and a brief description of their bases, where available, is provided. The values indicated have in general been subjected to rigorous peer review within the respective agencies.
For a number of the inorganics, which are required for proper nutrition, recommended daily intake values are also indicated.

3. Exposure Assessment

Exposure assessment is the qualitative and quantitative determination, or estimation, of the magnitude, frequency, duration and route of exposure of a particular physical, chemical or biological disturbance to the environment. It delineates the major pathways of exposure (e.g, air, water, food), the levels of exposure from each pathway, and the total exposure of the given population from all pathways that contribute to the health risk of concern. Data for exposure assessment may be obtained from monitoring studies of the contaminant and from dynamic modelling of its environmental fate.

Exposure assessment extends to an evaluation of the uncertainties associated with the determination or estimation. Individual exposures to single environmental media should be evaluated in the context of an integrated multimedia assessment of total exposure.

3.1 Soil Ingestion

Infants and young children ingest soil as a result of normal behavioural characteristics such as hand-to-mouth activity (i.e. finger licking and thumb sucking) and immature dietary habits (e.g. eating foods that have had direct contact with soil or dusts). The extent of such behaviours will vary between children and with age and therefore the amount of soil ingested will be variable.

The available scientifc information concerning soil ingestion rates is summarized in Appendix 1. Taking into account the available information, a value of 80 mg/day was selected as the soil intake rate for a child and 20 mg/day for adults. These assumptions are in keeping with the recommended Canadian Reference values for dirt,

dust and soil intake (HWC, 1988).

Estimation of intakes for each substance are based upon soil intakes rates and soil concentrations. Arithmetic mean concentrations are employed to estimate typical intakes for human receptors. This is a conservative measure, as the soil data are typically skewed to lower concentrations. Concentrations in the 0-5 cm surface layer are utillized as this is representative of the soil/dusts which receptors will have contact with. These levels are used provided they reflect the range of values measured at any of the three depths for the 1986 survey.

Average soil exposures are based on continual daily exposure. They are not adjusted for factors such as climate, time spent indoors and plausible residence time in the community. Adjustment for any or all of these factors would lower exposure estimates. Therefore, this model will tend to overestimate exposure. For contamination on recreational areas, exposures scenarios are adjusted for plausible time spent in the area.

Soil exposures are expressed as intake values where compared to reference doses, which are based on administered dose and not adjusted for absorption rates. Little information is available regarding the bioavailability of specific metals from soil. Intakes are adjusted for bioavailability from the soil matrix for specific metals. Where intakes are adjusted for biovailability from the soil medium, conservative values were selected.

3.2 Dermal Contact

Dermal contact with soil/dusts may occur for children through play and for adults through work and gardening. Dusts are present on essentially all surfaces (e.g., swings, pavement and buildings) and the amount of contact with the skin will depend on individual behaviour. For most inorganic forms of metals in solution, it is expected that dermal absorption will not occur to a significant extent (see Appendix III for a technical discussion). Inorganics that are soil-bound are expected to be dermally absorbed to a much lesser extent than those in solution. For example, bioavailability of 1% has in other cases been applied to metals in soil as an upper bound estimate. The bioavailability of inorganics on skin (percentage released from the soil matrix during skin contact), together with the limited penetration of the epidermis, suggest that this exposure will be of negligible magnitude. Discrete estimates of dermal uptake were therefore not undertaken. Metals for which exposures to high levels in occupational settings have been associated with contact dermatitis effects (chromium and nickel) are considered qualitatively.

3.3 Consumption of Homegrown Garden Produce

Those individuals who grow and consume vegetables or crops grown on

residential properties may have additional intakes by this indirect exposure pathway. Levels of inorganics in garden produce from the areas in question have not been sampled. It is therefore not possible to provide a definitive analysis as to the potential intake of these inorganics from fruits and/or vegetables that may be grown in residential gardens in the area. The rate and extent of uptake is influenced by the type of produce, length of growing season and soil characteristics. Reliable estimation of individual intakes would therefore require direct sampling data on vegetables.

In order to examine the question of intake from homegrown produce, concentrations of contaminants in produce are modelled based on bioavailability or assumed based on reported critical tissue concentrations; the latter is the limiting factor. This is combined with assumptions regarding adult and child consumption of these foodstuffs to yield crude estimates of intake. These calculations are described in Appendix II. These values are very crude estimates and are therefore are not included in the calculations of total daily intake. They are utilized to provide (1) an indication that individuals may have increased intakes if consuming homegrown produce and (2) for gross comparison to other exposure pathway intakes.

4. Risk Characterization

In this step, estimates of exposures from single or combined pathways are compared with the information on dose-response and current exposure limits defined from toxicological information. This step includes a determination of the major routes of exposure and of the specific population or part of the environment at risk. The magnitude and type of risk from each route of exposure are assessed, and an evaluation is made of the contribution of the particular route to the overall risk.

Outcomes can include the probability that a particular adverse effect will occur in a given population, the relative contributions of various pathways to outcomes and qualitative judgements on the potential for health effects to occur. Where an inorganic may be an essential dietary nutrient, such information is also taken into account.

The output of risk characterization is usually given as a single point estimate of risk. However, it must be realized that some degree of uncertainty is contained in these apparently precise predictions of risk.

The question of uncertainty in risk assessment has been reviewed (Finkel, 1990). The sources of uncertainty include

- lack of precision of scientific measurements;
- incomplete knowledge of underlying biological and environmental processes;

- variability in human and animal populations;
- assumptions and limitations inherent in predictive models; and
- random workings of chance.

Time limitation precluded systematic quantification of the attendant uncertainties of each stage of the risk assessment process. However, at each decision point, assumptions were conservative. This does not mean that worst-case assumptions were always accepted.

1 ARSENIC

1.1 HAZARD IDENTIFICATION

People are chronically exposed to low levels of inorganic and organic arsenic in the environment. Because these exposures can occur by a number of different pathways, this substance is considered to be a multimedia contaminant. Available evidence shows that organic arsenicals in the environment, such as those found in fish and other foods, are absorbed after ingestion but have very low toxicity to man and are excreted unchanged. These organic compounds are, on the whole, poorly characterized. There is considerable evidence, both from man and from other organisms, of the toxic effects of inorganic arsenic, which is present as compounds of either the AsIII or AsV form. There is, however, little information describing the inorganic compounds to which humans are exposed to environmentally. This is especially true of inorganic arsenic in food.

1.1.1 Absorption and Metabolism

The fraction of inorganic arsenic absorbed from the gastro-intestinal tract will vary, depending on the chemical species, solubility, dose administered and the matrices of the compounds administered. In general, it is expected that arsenic in aqueous solutions would be better absorbed than arsenic bound to particulate matter (U.S. EPA, 1984b). Arsenic in soil will more likely be adsorbed to particulate matter and not be in solution. Therefore, the bioavailability of arsenic from soil is predicted to be much less than that of inorganic arsenic in solution.

With respect to inorganic arsenic that is ingested, the following observations can be made concerning its absorption from the human digestive tract. About 90% of the inorganic arsenic in food is absorbed in the gastro-intestinal tract. From water, arsenic is presumed to be totally available.

In the case of the soil matrix, the composition of arsenic in soil and dusts is not well characterized. Information available in the literature suggests that 3.5% to 45% of the arsenic in the soil is extractable by mild acid. This fraction could be utilized as a very crude surrogate of bioavailability of the arsenic in ingested soils.

The relative intake of arsenic by inhalation is very small. Airborne arsenic is mainly in the form of suspended particulates, the chemical nature of which is not well characterized, although it is usually assumed to be inorganic. It is assumed that 30% of the inhaled arsenic is deposited and absorbed in the respiratory tract. Most of the remaining 70% of the inhaled arsenic is swallowed, and up to 50% of the swallowed arsenic is absorbed in the

gastro-intestinal tract. Therefore, up to 65% of the particulate arsenic inhaled is absorbed. The slow absorption and clearance of some of the insoluble particulate arsenic leads to a build-up in the lungs. Thus, arsenic is retained even after exposure has ceased.

Dermal absorption of dissolved arsenic compounds may occur, but is considered minimal, and therefore this route of environmental exposure would be considered inconsequential, as skin is rather impermeable to water and dissolved ions (Scheupler and Blackwell, 1971).

Once absorbed, arsenic is transported to other tissues of the body by the red blood cells and the blood plasma. The liver detoxifies the inorganic arsenic by methylating it to dimethyl arsenic acid. The excretion of the absorbed arsenic is largely through the urine. Fecal arsenic represents ingested insoluble particulates. Total intake and excretion, when averaged over a week, are in balance. Intake and excretion vary with age and sex from about 5 to 30 µg/day on average in Canada.

1.1.2 Toxicology

Some question exists as to whether arsenic is a nutritionally essential dietary element, and this has been the subject of previous scientific review (U.S EPA, 1988). Studies with chickens and small mammals fed a diet almost free of inorganic arsenic suggest that a certain daily dose of arsenic is needed for normal development and health of the animals. There is no direct evidence that arsenic is an essential element for humans. Marcus and Rispin (1988) have suggested that the nutritional requirement for animals for arsenic lies between 12 and 50 µg/day. This is based on research work using chickens, goats and rats fed on arsenic-deficient diet. It is considered plausible that there is a nutritional requirement for arsenic in humans, although no arsenic deficiency disease in humans has been reported.

The toxicological aspects of inorganic arsenic has been reviewed in detail in a number of reports (WHO, 1981; U.S. EPA, 1984b; U.S. EPA, 1988b; MOE, 1991b). Arsenic has a number of known toxic effects in humans. Occupational exposure to arsenic dust and ingestion of water with high concentrations leads to irritation of mucous membranes, hyperpigmentation and hyperkeratosis, and neurotoxic and cardiovascular effects.

Arsenic is a potent teratogen in animals, producing a wide spectrum of malformations. The effective teratogenic dose is near the maternal toxic dose. The few studies in humans have produced inconclusive results.

Arsenic induces sister chromatid exchange (SCE) and chromosome aberrations <u>in vitro</u> but does not cause gene mutations. It does not

directly affect DNA but rather appears to inhibit DNA repair processes. Both AsIII and AsV are co-mutagenic and inhibit the growth of cells in vitro. AsIII is about ten times more potent than AsV.

Although arsenic does not appear to induce chromosome aberrations in vivo in experimental animals, several studies suggest that humans exposed to high levels have higher frequencies of SCE and chromosomal aberrations in peripheral lymphocytes.

The toxic effects of arsenic - on the myocardium and the vascular, neurological, dermal, haematological, hepatic and renal systems - all occur at doses higher than the dose correlated with the induction of carcinogenicity. The most significant toxicological endpoint, in terms of evaluating the health risk related to potential exposures to arsenic soil contamination, is skin cancer.

The International Agency for Research on Cancer (IARC) states that there is "sufficient evidence that inorganic arsenic compounds are skin and lung carcinogens in humans. The data suggesting an increased risk for cancer at other sites are inadequate for evaluation" (IARC, 1980). Animal studies generally have not shown increased rates of tumour formation. The only positive studies involve particulate arsenic that has been instilled intratracheally in hamsters.

There is clear evidence that workers exposed to occupational levels of arsenic dust through inhalation in non-ferrous smelters have increased rates of lung cancer (U.S. EPA, 1984b). Studies from Taiwan and Mexico show a strong link between arsenic in drinking water and skin cancer. Studies in the USA have not found a similar relationship. This is ascribed to lower exposures and possibly increased sensitivity to arsenic in the Taiwanese and Mexicans because of a poorer diet. There is a suggestion in the Taiwanese studies of a linkage between ingested arsenic and internal cancers.

The mechanism by which arsenic induces cancer is unknown.

1.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

There is a lack of international consensus on the carcinogenic risks to man posed by oral exposure. The U.S. EPA considers arsenic as a carcinogen without a threshold value; the World Health Organization assumes a threshold level for oral exposure. The WHO has developed a provisional maximally permissible oral dose of 2 µg/kg/day of inorganic arsenic, based on drinking water consumption. This is roughly equivalent to 140 µg/day for an adult.

The Ontario drinking water objective has recently been revised to 25 µg/L (G. Jenkins, personal communication). U.S. EPA has set an Interim Maximum Contaminant Level of 50 µg/L (U.S. EPA, 1991). The proposed health advisories for one day to lifetime exposures are

also 50 µg/L. At this concentration, adverse effects would not be expected to occur, and a margin of safety to protect sensitive members of the population is included.

EPA states that the maximum likelihood estimate of lifetime risk of skin cancer (not of dying from skin cancer) for a 70 kg person consuming 2 L per day of water contaminated with 1 µg/L of arsenic (equivalent to an intake of 2 µg/day) is 3×10^{-5} to 7×10^{-5}. This estimate is based on the Taiwanese studies of skin cancer incidence. U.S. EPA has recommended utilization of a risk level of 5×10^{-5} $(\mu g/L)^{-1}$.

There is experimental evidence that a diet poor in protein and calories in animals leads to a higher sensitivity to the effects of inorganic arsenic because the amount of methyl donors in the liver is decreased. Since the diet of the Taiwanese is deficient in protein and calories, they may well be more sensitive to the effects of inorganic arsenic. Therefore, the risk of cancer based on the Taiwanese studies may be overestimated if applied to Western populations like Canada. That is, the U.S. EPA risks may be overestimates.[2]

The argument has also been advanced that the detoxification (methylation) pathway for inorganic arsenic becomes saturated at around 250-500 µg/day (Marcus and Rispin, 1988). Exposures above this saturation level are said to lead to an increase of inorganic arsenic in the blood and hence cancer, especially skin cancer. The authors concluded that arsenic is a threshold carcinogen requiring a daily intake of more than 200-250 µg to have an effect. However, although the evidence shows that there is a threshold for methylation, a threshold for cancer has not been demonstrated, as the amount of inorganic arsenic excreted is linear with dose.

The ambient air quality criterion in Ontario is 0.3 $\mu g/m^3$ (24 hr average) and the point-of-impingement value is 1 $\mu g/m^3$ (0.5 hr average).

U.S. EPA estimates that the lifetime cancer risk from arsenic in air at a level of 1 $\mu g/m^3$ is 4.29×10^{-3} (U.S EPA, 1991). This estimate is based on occupational exposures in copper smelters in the USA.

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[2]In a recent memorandum by the administrator of the U.S. EPA, it is recommended that the 5×10^{-5} µg/L unit risk level be adopted. The memorandum counsels "In reaching risk management decisions in a specific situation, risk managers must recognize and consider the qualities and uncertainties of risk estimates. The uncertainties associated with ingested inorganic arsenic are such that estimates could be modified downwards as much as an order of magnitude, relative to risk estimates associated with most other carcinogens."

1.3 HUMAN EXPOSURE ASSESSMENT

The arsenic ions form insoluble salts with a number of cations in soils and are adsorbed by soil constituents, such as organic colloids and iron and aluminum oxides. Arsenic is held quite strongly by soils, especially fine-textured ones, and is leached very slowly. In reducing environments, the predominant species is AsIII, whereas AsV predominates in oxidizing environments. Microbes can convert inorganic arsenic to volatile methylated arsenicals.

The reported amounts of extractable arsenic range from a low of <5% to as much as 60+%, depending on the soil type and the extracting agent used (Woolson et al., 1971.; Tetratech, 1985).

The most common forms in water are anions of AsIII and AsV. AsIII forms are found only in strongly reducing environments, such as anoxic bottom waters or sediments. Arsenic is adsorbed on hydrous iron III oxides and clays and eventually sediments out.

1.3.1 Estimated Intakes from Individual Sources

1.3.1.1 Soil and Dust

From the data on total arsenic concentrations (µg/g, dry weight) for 1986 and 1987 in the 0-5 cm layer in soil at Port Hope, the following statistics can be calculated:

- average concentration: 20 µg/g (n=76)
- minimum concentration: 2 µg/g
- maximum concentration: 234 µg/g

The maximum concentration represents only one site (site #7), a small area adjacent to the railway station. The majority of reported levels in 1986 and 1987 are less than 20 µg/g total arsenic. The SYMAP predicted contours indicate a range of levels from <10 µg/g to 50 µg/g over most of Port Hope; levels greater than 50 µg/g are confined to two small areas. Individual site measurements of greater than 100 µg/g arsenic were seen at sites #7 and #59 (site #59 is a residential garden soil).

Using the soil consumption assumptions and the concentrations, the intake of total arsenic (µg/day) in the one-to-six year age group and for adults are calculated in Table 2. The maximum intake associated with the maximum reported concentration is an overestimate because the site in question (site #7) is not an area where an individual would spend more than a small amount of time. Actual exposures in this area adjacent to the railway station are likely to occur only intermittently. It is assumed in these calculations that the soil arsenic concentrations are representative of the concentrations in the ingested dust and dirt.

There is no information available on the speciation of arsenic in

the soil at Port Hope. The predicted range of arsenic (3.5-45%) that is bioavailable from soil and can be absorbed in the gastro-intestinal tract is applied to these total intakes. The corresponding ranges of uptake corresponding to the calculated intakes are shown in Table 2 in parenthesis.

It is assumed here that all of the absorbed arsenic is inorganic.

TABLE 2 ESTIMATED INTAKES OF TOTAL AND ABSORBED INORGANIC ARSENIC FROM SOIL/DUST INGESTION

	Child (1-6 years)	Adult
Soil Concentration Range Average	2 - 234 µg/g 20 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake (Absorbed)	1.6 µg/day (0.06-0.7)	0.4 µg/day (0.02-0.2)
Minimum Intake (Absorbed)	0.16 µg/day (0.006-0.07)	0.05 µg/day (0.002-0.02)
Maximum Intake (Absorbed)	18.7 µg/day (0.65-8.4)	4.7 µg/day (0.16-2.1)

1.3.1.2 Air

The concentration of arsenic in the air at Port Hope is not specifically known. The measured concentration in urban air in Ontario averages 1-2 ng/m^3 (MOE, 1988). A value of 1.5 ng/m^3 will be assumed as representative of urban air for Port Hope. The amount inhaled ranges from 8 ng/day for children to around 44 ng/day for adults. The amount absorbed ranges from 3 to 30 ng/day, respectively, assuming 30% absorption in the lungs and 35% in the gastro-intestinal tract.

1.3.1.3 Drinking Water

No measured levels of arsenic in drinking water were available from this community. According to the Drinking Water Surveillance Program (MOE, 1988), no arsenic was detected in samples of raw or treated tap water taken at the Oshawa's water treatment plant in 1987 (the detection limit is 1 µg/L). The average concentration of arsenic in Lake Ontario is about 0.5 µg/L. This amount is used to obtain a rough estimate exposure from water. The estimated intakes

by this route varies from 0.3 µg/day for children to 0.8 µg/day for adults.

1.3.1.4 Food

MOE (1991) has calculated (1) the intake of total arsenic and (2) the amount of inorganic arsenic absorbed (to the nearest µg/day) from food by various age groups for general urban Ontario populations. These values (to the nearest µg/day) are, respectively:

• 1 year old	9	5
• child 1 to 11 years	13	7
• male adolescent	24	13
• female adolescent	15	8
• male adult (>21 yr)	21	12
• female adult (>21 yr)	14	8

It is apparent that dietary exposure to arsenic varies with age and sex. It varies from about 5 µg/day for 1-year-old children to 12.6 µg/day for adolescent males. For this assessment, the values of 13 µg/day intake of total arsenic and 7 µg/day for inorganic arsenic absorbed are assumed for children, choosing the upper end of the range. The values used are the averages for males and females: for adults 17.5 µg/day intake and 10 µg/day absorbed. These values allow for the presence-and 90% absorption-of inorganic arsenic in food.

1.4 RISK CHARACTERIZATION

The integrated exposure estimates and assumptions employed for typical adults and children are described in Table 3. The scarcity of data on water and air concentrations limits the value of these estimates. The intake from inhalation, which is estimated at low nanogram quantities for Ontario, is extremely small. The estimated intakes from food are by far the largest contributor to arsenic exposures for both adults and children. The estimated intakes and amounts absorbed from soil/dusts represent only a relatively small fraction of total intakes. The relative contribution of each of the pathways is shown in Table 4.

The dermal uptake of dissolved arsenic is considered to be very low, and therefore this pathway of exposure is not of consequence.

The background intake of arsenic in Ontario from ingestion and inhalation, including the intakes for various age groups living in urban areas has been examined (MOE, 1991b). Using an average concentration of arsenic in Ontario soils of 6 µg/g (Frank et al., 1984), the resultant intake was found to be quite low, about 0.3 to 0.6 µg/day for total arsenic and 0.15-0.3 µg/day for inorganic arsenic. Exposure estimates for food and drinking water in the current assessment are based upon these arsenic intake figures for

typical urban populations in Ontario. These background intakes provide a useful point of comparison against which to assess the discrete exposure and associated risk level which might be encountered from ingestion of the soils/dusts in Port Hope. For the general population, food is the major source of intake, accounting for approximately 90% of the total exposures.

As indicated above, the typical Ontario child ingests, on an average, a background intake of about 13 µg/day total arsenic, or 7 µg/day absorbed inorganic arsenic, from food and water. Arsenic exposure from soil and dust ingestion for the general urban population of children has previously been estimated to contribute only 5% (MOE, 1991b). The calculated average intake of arsenic from soil/dust in Port Hope (1.6 µg/day total, 0.06-0.7 µg/day inorganic arsenic) would increase this total intake and absorbed inorganic arsenic by relatively small fractions (15% for intake and 0.8-9.5% for inorganic arsenic). Therefore soil/dust ingestion at the average measured concentrations in the Port Hope survey (20 µg/g) would increase the estimated intakes by no more than 15% and absorbed amounts by no more than roughly 10%.

Typical Ontario age groups older than 6 years ingest roughly 18.2 µg/day of total arsenic, and absorb about 11 µg/day of inorganic arsenic from food and water. For this age group, the arsenic in soil and dirt increases the average intake of total arsenic by 2% and of absorbed arsenic by 0.2-2%. This is a smaller fractional increased exposure than estimated for children.

According to the SYMAP predicted soil levels, the level of arsenic in Port Hope ranges from <10 to 50 µg/g over most of the city and >50 µg/g in two small areas. If it is assumed that the relationship between soil level and inorganic arsenic holds, then at exposure to 50 µg/g the absorbed arsenic intake would increase by about 2-25% for children and 0.5-5% for adults.

In a recent study of populations living in the vicinity of a copper smelters, arsenic in urine was used as a measure of exposure to soil, household dust and air particulates (Polissar et al., 1990; Binder et al., 1987). The analyses showed that household settled dust had a lower concentration of arsenic than the soil, and that indoor particulate had a lower concentration than outdoor. Only children younger than six and living within one-half mile of one of the smelters had urine concentrations higher than the controls. The authors conclude that hand-to-mouth activity is the primary pathways of these exposures and that mean soil arsenic levels of <100 µg/g are not associated with excess exposure in young children.

The assessment of risk for arsenic is approached here utilizing calculations based upon (1) estimates of population skin cancers, (2) individual incremental cancer risk estimates and (3) comparison of exposure estimates to possible toxicity thresholds. The first

two considerations stem from the hypothesis that arsenic is a non-threshold carcinogen and the latter from the possibility that there is a threshold dose for arsenic-induced skin cancer.

1.4.1 Non-Threshold Risk Consideration

A commonly hypothesized mechanism for carcinogenesis presumes that no threshold or level of exposure to a chemical exists that does not pose a finite probability of being carcinogenic. Such hypothesis underlies the guidelines currently utilized in the U.S. EPA and other agencies for the assessment of cancer risk. Risk estimates based upon unit risk level for oral exposure to arsenic as derived by U.S. EPA are presented below. Assessment for population and individual health risk are discussed.

One manner of estimating cancer risks is in terms of expected cancer frequency per population. On the basis of the U.S. EPA risk estimates (U.S. EPA, 1988b; Brown et al., 1989), it has been evaluated elsewhere (MOE, 1991b) that the predicted number of cases of skin cancer per year in all of Ontario caused by exposure to arsenic in drinking water is <0.5-2.1, depending on the water consumption values utilized. The amount of inorganic arsenic absorbed by an adult in Ontario from all sources is about 10 µg/day, or about 12.5 to 50 times that from water alone. On this basis, the expected number of skin cancer cases per year in all of Ontario would be <25. The predicted number of cases for all of Ontario caused by the ingestion of soil/dust at background exposure rates is 0.2-0.5/year. As was discussed above, the U.S. EPA risk estimates and hence these predicted number of skin cancer cases may well be overestimates.

The actual number of new skin cancer cases in Ontario is around 5,000 per year (MOE, 1991b). Thus, the frequency of cases of skin cancer caused by arsenic in the entire province would be estimated at about 0.5% of the total.

The frequency of expected skin cancers within a smaller urban population such as Port Hope can be calculated similarly. The expected frequency of skin cancer for a population of 10,000 exposed to 10 µg/day of absorbed inorganic arsenic in food and water alone over a lifetime would be 0.04/year. The incremental increase in skin cancer frequency from additional arsenic uptakes (approx. 0.2 µg/day) from soil/dust ingestion at average arsenic soil concentrations over 70 years would be 0.0006/year, or 0.05 skin cancers in a lifetime. The maximum arsenic concentration measured in the survey was 234 µg/g, or roughly 12 X the average. For comparative purposes, the corresponding increase in risk level for a maximally exposed population is therefore 12 X 0.0006 = 0.0072 cases/year. These calculations assume that the entire population would be exposed to these levels over a lifetime, which is not a plausible exposure scenario and therefore these figures are likely to overestimate risk on this basis alone.

TABLE 3 ESTIMATED DAILY INTAKE OF ARSENIC – INTEGRATION OF EXPOSURE PATHWAYS

Substrate or Medium	Child (1–6 years old)	ADULT
SOIL/DUST Average Concentration	20 µg/g	20 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake from Soil	1.6 µg/day	0.4 µg/day
Average Absorbed Intake	0.06 – 0.7 µg/day	0.02 – 0.2 µg/day
AIR Average Air Concentration of Port Hope	not known	not known
Average Air Concentration, Urban Air	1.5 ng/m^3	1.5 ng/m^3
Volume Inhaled	5 m^3/day	22 m^3/day
Estimated Inhaled Intake	7.5 ng/day	33 ng/day
DRINKING WATER Assumed Tap Water Concentration	0.5 µg/L	0.5 µg/L
Daily Water Consumption	0.6 L/day	1.5 L/day
Estimated Intake	0.3 µg/day	0.8 µg/day
FOOD Total Intake	13 µg/day	18 µg/day
Estimated Absorbed from Food	7 µg/day	10 µg/day
ESTIMATED TOTAL DAILY INTAKE	≈ 15 µg/day	≈ 19 µg/day
ESTIMATED AMOUNT ABSORBED	7.4 – 8 µg/day	10.8 – 11 µg/day

TABLE 4 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate or Medium	Percentage of Total	
	Child (1-6 years old)	Adult
Soil/Dust	9	2
Air	0	0
Drinking Water	4	7
Food	87	91
TOTAL	100%	100%

For non-threshold carcinogens, risks may also be estimated as the incremental probability of an individual developing a particular cancer over a lifetime as a result of exposure to a potential carcinogen. This would represent the incremental or excess individual lifetime cancer risk. The U.S. risk factor for drinking water of 5×10^{-5} (at a 2 µg/day intake) is utilized. If we assume that the average chronic daily intake from soil/dust is 0.016 µg/kg/day and adjust for absorption from soil utilizing a conservative estimate (45%), the incremental lifetime cancer risk is estimated at 1×10^{-5} or less (as shown in Table 5). At an exposure concentration of 50-100 µg/g, the corresponding risk level would be 2.5×10^{-5} - 5×10^{-5}. These values are in the order of magnitude of risk generally considered to be negligible. Also, as indicated in the IRIS database (U.S. EPA, 1991), the U.S. EPA administrator in adopting the unit risk levels has counselled that the qualities and uncertainties associated with ingested inorganic arsenic are such that risk estimates could be modified downward by as much as one order of magnitude. This has not been done in this case, however it does further suggest that the values very probably overestimate actual risk.

To place these risk values in perspective, it is useful to compare them to the estimated risk level for typical urban populations in Ontario for food. As indicated above, for groups other than young children, the food intake ingestion figure is approximately 10 µg/day, which, if applied to the cancer risk factor, would yield an incremental risk of approximately 2.5×10^{-4}. The relative risk for these pathways is illustrated in Figure 2. As shown, the predicted risk level associated with average exposure to arsenic in soil is only a fraction of that associated with everyday food exposures.

1.4.2 Threshold Considerations

As a corollary to the assessment of risk utilizing non-threshold assumptions, it is useful to examine the estimated intakes of inorganic arsenic from these soil/dust levels in relation to the detoxification threshold in the mechanistic model proposed by Marcus and Rispin (1988). The authors' system describes an intake of 200-250 µg/day before an effect would occur, and the level of enzyme saturation is in the range of 500 µg/day. The soil/dust exposure estimate for Port Hope at the maximum soil concentration is 0.4-8.4 µg/day, which is >20-fold less than this proposed threshold for carcinogenic effects. Total intakes from all sources would be well within the range of normal metabolic detoxification shown by studies of arsenic metabolism. This approach would suggest that exposures to the arsenic in the soils/dust in question do not have adverse consequences.

Comparisons to allowable levels of arsenic in drinking water can also be made. Based on the Ministry of the Environment (MOE) drinking water objective of 25 µg/L and a consumption of 1.5 L/day for adults, the inorganic arsenic limit would be roughly 37.5 µg/day. The estimated intakes and absorbed amounts in total from all pathways including soil/dusts are well below this value. Similarly, discrete soil and total intakes are several fold lower than the WHO provisional maximum daily intake for inorganic arsenic.

SUMMARY

- Inorganic arsenic when ingested is a human carcinogen resulting in an increased incidence of skin cancer. Arsenic may or may not be an essential dietary element.

- Dermal contact and absorption of arsenic from soil is qualitatively considered to be an insignificant pathway of exposure owing to the very limited absorbability of arsenic through the skin.

- With respect to the exposure estimates for Port Hope, normal intakes of arsenic are predominantly through the food pathway, accounting for approximately 90% of daily exposure. The average measured arsenic level derived from all survey sites is approximately 20 µg/g. The majority of measurements are below this value. The estimated average daily intake of inorganic arsenic from soils/dusts at this concentration is predicted to result in increases of absorbed arsenic intakes of 0.8-9.5% for children and 0.2-2% for adults. Contamination at 50 µg/g would increase intakes by about 0.5-25% over background intake.

- The incremental average absorbed intake for children is 0.06-0.7 µg/day and for adults is 0.4 µg/day. The maximum estimate based upon the highest measured concentration is 0.6 to 8 µg/day for children and 0.16 to 2.1 µg/day for adults. As the sites associated with the highest levels of soil arsenic are non-residential, actual exposures are likely much smaller than suggested by these values as time spent in these locations would not be continual.

- Based on U.S. EPA skin cancer potency factor for ingested arsenic, average daily intakes from soil and dust, assuming daily exposure for 70 years, would predict an increased frequency of skin cancer of 0.0006 per year (0.05 over lifetime) for a hypothetical population of 10,000 (the approximate population of Port Hope). Based on the highest measured concentration, the corresponding increased risk is 0.0072 cases per year.

- Calculated average individual incremental risk from soil/dust is estimated at 1×10^{-5} for chronic daily intake over a lifetime. This is more than 10 X lower than the risk level associated with daily exposure in diet, similarly calculated. Risk levels corresponding to 50 µg/g and 100 µg/g soil arsenic are 2.5×10^{-5} and 5×10^{-5}.

- The total intake (child and adult) is well within the range of normal metabolic detoxification as suggested by the Marcus-Rispin model. It is also well below the oral intakes allowable under the MOE drinking water objective for arsenic and the WHO maximum permissible intakes for inorganic arsenic.

- The assessment provided is conservative in that:

  - the maximum likelihood estimates based on the Taiwan studies may overestimate the cancer potency slope and therefore the estimated risk for the reasons given above.

  - average soil exposures are based on year-round and lifetime exposures. They are not adjusted for factors such as climate, indoor/outdoor activity and plausible residence time in the community. Adjustment for any or all of these factors would lower exposure estimates.

  - the upper end of the range of estimates of bioavailability of inorganic arsenic from the soil matrix is utilized.

TABLE 5 CANCER RISK ESTIMATES FOR INORGANIC ARSENIC IN SOIL/DUSTS

	Chronic Daily Intake[1] (µg/kg/day)	CDI[2] Adjust for Absorption (µg/kg/day)	Type of Cancer	Weight of Evidence[3]	Slope Factor[4] (µg/kg/day)	Pathway[5] Risk Level
Soil/Dust Ingestion	0.016	0.0072	skin	A	1.5×10^{-3}	1×10^{-5}
Food		0.175	skin	A	1.5×10^{-3}	2×10^{-4}

1. The chronic daily intake is the estimated intake from that pathway daily as averaged over a lifetime and adjusted for body weight.
In this case, $\text{CDI} = \frac{\text{intake(child) x 7 years}}{\text{15 kg x 70 years}} + \frac{\text{intake(adult) x 63 years}}{\text{70 kg x 70 years}}$

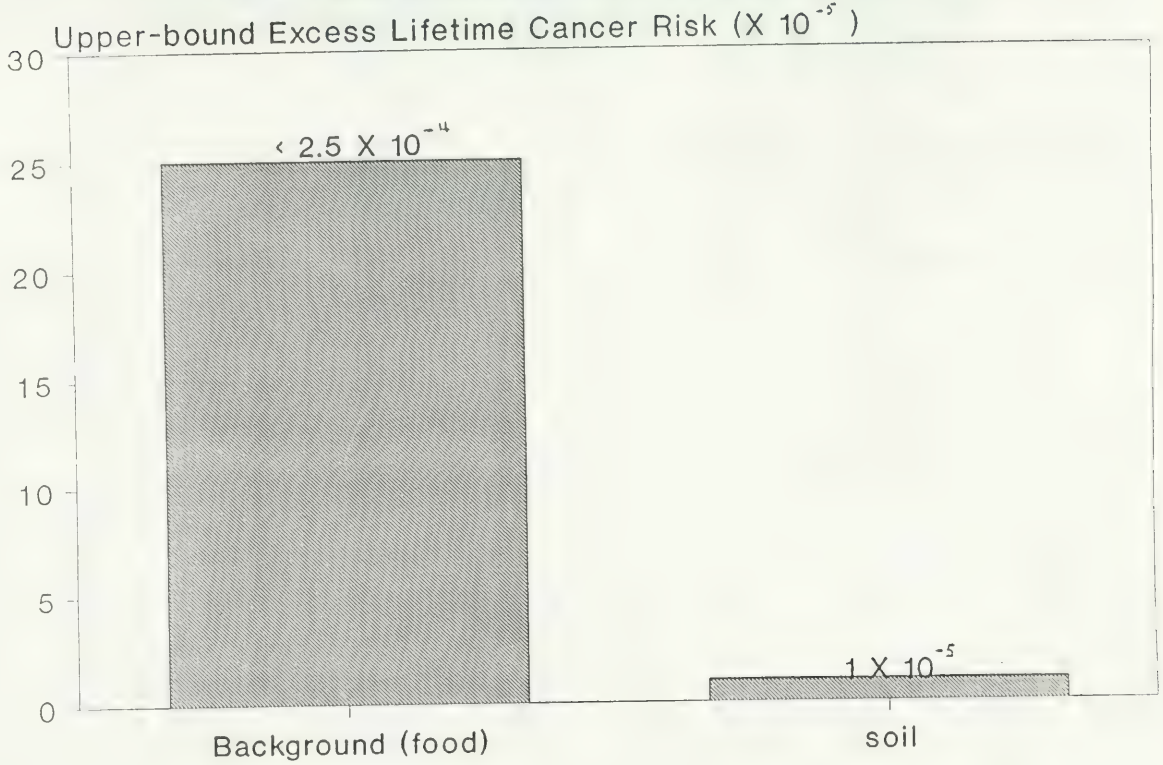
2. Where absorption from the media ingestion is significantly different from that which the cancer slope factor is derived, the CDI is adjusted for absorption. Bioavailability from soil is assumed here to be 45%.

3. U.S. EPA weight of Evidence classification (U.S. EPA, 1991).

4. Slope factor is the maximum likelihood estimate of skin cancer risk due to 1 µg/kg/day of arsenic intake, based on U.S.EPA cancer potency for drinking water of 5×10^{-5} $(\mu g/L)^{-1}$. The slope factor is based on dose administered in drinking water and an assumed 100% absorption. These risk estimates are based on a dose-response model that assumes linearity at low doses and would overestimate risk of risk decreases faster than linear or if a threshold exists for arsenic-induced skin cancer (U.S.EPA, 1988).

5. Cancer risk estimates are expressed as one significant figure only.

Figure 2. Relative Incremental Cancer Risk Estimates for Arsenic in Soil



2 ANTIMONY

Antimony, which is a semi-metal belongs to the same periodic table group (Sb) as arsenic and shares with it many physical and chemical similarities. However, antimony is considered much less toxic than arsenic. Antimony is found primarily in two oxidation states, trivalent or pentavalent, and can form organic or inorganic complexes.

Antimony occurs usually as sulfides or oxides, and sometimes as native metal. It is frequently found as a by-product of lead refining. It is commonly used in alloys to harden other metals such as lead, copper or tin. Trioxide compounds are used as flame retardants in textiles and plastics. Some antimonial compounds, such as antimony potassium tartrate (tartar emetic) and sodium stibogluconate, are used therapeutically to treat parasitic infections and tropical diseases. Formerly, antimony compounds were used as emetics in humans; the usual dose was 30-50 mg in an aqueous solution. Antimony is considered a non-essential element with no observed bodily metabolic function.

2.1 HAZARD IDENTIFICATION

2.1.1 Absorption and Metabolism

The metabolism and distribution of antimony in the body are dependent upon the route of exposure and the valence of the compound. Trivalent antimony is more readily absorbed than pentavalent forms.

The absorption of antimony from the gastro-intestinal tract is low, and most ingested antimony compounds are excreted in the feces. Felicetti et al. (1974b), in a study involving the administration by gavage of radio-labelled antimony compounds to Syrian hamsters, concluded that "very little" of these compounds were absorbed from the gastro-intestinal tract, probably less than 1%. These forms were the relatively insoluble oxides; water-soluble organic forms may be more readily absorbed. Elinder and Friberg (1986) report absorption of at least 15% for ingested antimony potassium tartrate in study involving a single oral dose to mice. Although the chemical species of antimony in the soil surveys was not determined, the above information and the likelihood that the predominant form of antimony in soils is the oxides would suggest that 1% is a reasonable bioavailability factor for gastro-intestinal absorption. A somewhat more conservative value of 5% will be utilized for the current assessment.

There is very little information pertaining to absorption of antimony following inhalation exposures. A small number of studies have shown that antimony is absorbed from the lungs (Felicetti et al., 1974 a, b). A study cited in Reference Man (ICRP, 1975) indicates a 95% absorption rate from lungs for inhaled antimony,

but no further information is provided. In essence, there is no definitive quantitative information for the pulmonary bioavailability of antimony and any value utilized would be arbitrary.

During absorption, trivalent antimony readily binds to erythrocytes in the blood, where it impedes haemoglobin function. It is primarily excreted as bile into the gastro-intestinal tract and then eliminated in the feces. Pentavalent antimony does not have such a high affinity for cells and remains in the plasma, from where it is excreted in the urine. Although the route of exposure influences the distribution, antimony is found primarily in the kidney, liver and thyroid. Inhaled antimony can remain in the lungs for a long time. Antimony is also stored in bone, teeth and hair.

2.1.2 Toxicology

The limited toxicological information on antimony and its compounds has been reviewed previously (U.S. EPA 1985b, 1987, 1991; Stokinger, Elinder and Friberg, 1986). The most relevant information is summarized below. Antimony and its compounds vary in their toxicity but are generally regarded as having a high order of toxicity and the capability of affecting several organ systems. For example, rodents exposed to fumes of antimony oxide have developed pneumonitis, fatty change of the liver, decreased leukocyte count and damage to heart muscle. Like arsenic, the substance is more toxic in the trivalent than in the pentavalent form.

Acute high-level exposures have been reported to cause nausea, vomiting, abdominal pain, diarrhea and dehydration. They are also associated with cardiovascular effects and irritation of mucous membranes and skin (antimony spots). Cardiac degeneration is one of the most serious human health effects associated with antimony exposure. These symptoms of exposure are generally found in individuals exposed in the workplace to high concentrations in air and dusts.

A number of side effects have been associated with tartar emetic use, such as liver disease, alterations in ECG and in some cases, death. There are few reported cases of acute poisoning in humans. One case of a contaminated lemon drink (approximately 30 mg/L) produced acute poisoning in 150 children.

With respect to skin exposure, "antimony spots" are transient skin pustules occurring in people working with antimony compounds. One study in an antimony smelter found that 62.7% of examined workers had evidence of "antimony dermatosis."

According to U.S. EPA (1980), multimedia exposures are essentially negligible by comparison to occupational exposures at which discrete clinical health effects have been observed. The most significant and best characterized effects associated with these

high occupational exposures is myocardial damage (U.S. EPA, 1991).

The carcinogenicity of antimonial compounds has not been proven for humans. It is not well supported by occupational health studies in great Britain and the U.S.A.; such studies generally involve past exposures to high levels of antimony, often associated with exposure to arsenic and other substances. The commonest form of antimony to which workers are exposed is antimony trioxide. In Great Britain, the production of antimony trioxide was reported to be associated with an increased incidence of lung cancer but the finding is questionable and unconfirmed. In the U.S.A. there were no associated cancer cases found in exposed workers. The American Conference of Government Industrial Hygienists (ACGIH) has concluded that antimony trioxide is a chemical associated with industrial processes which are suspected of inducing cancer.

The U.S. EPA has not evaluated the human carcinogenicity of antimony. ACGIH (1986) regards antimony oxide as a suspected carcinogen in humans. IARC (1990) has concluded that there is inadequate evidence for the carcinogenicity of antimony trioxide and antimony trisulfide in humans, but that there is sufficient evidence in experimental animals. Antimony trioxide is ranked as possibly carcinogenic to humans (group 2B). One study in rats found a high frequency of lung neoplasias after exposure to 1.6 and 4.2 mg Sb/m^3 for one year (Watt, 1983). No significant increase in the incidence of tumours in rats and mice was found after exposure to 5 mg/L antimony as potassium antimony tartrate in drinking water (Schroeder et al., 1970). However, the methodology of this last study could only detect visible tumours at necropsy and was not a complete histopathological examination.

No genetic or related effects in humans were reported.

2.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

Very few studies of the chronic oral toxicity of antimony compounds were found in the literature. There are lifetime studies with rats (Schroeder et al., 1970) and mice (Kanisawa and Schroeder, 1969) in which drinking water containing 5 µg/g antimony in potassium antimony tartrate was administered. In the rat study, both sexes exhibited decreased longevity, and altered cholesterol and glucose levels were observed in male rats. These effects did not appear until later life. No increase in tumours or effects on heart weight were observed. Decreases in life span were also noted in the mice studies, although the degree of toxicity was less severe in the mice than in the rats.

The U.S. EPA has developed an oral reference dose of 0.0004 mg/kg/day by applying an uncertainty factor of 1,000 to the lowest observed adverse effect level (LOAEL) of 0.35 mg/kg bw/day in the Schroeder et al. study (U.S. EPA, 1991). This is equivalent to an oral intake of 28 µg/day for a 70 kg adult, or 6.0 µg/day for a 15

kg child, assuming 100% absorption from the drinking water. The applied uncertainty factor was calculated following: a factor of 10 for interspecies conversion, 10 for protection of sensitive individuals, and 10 because a NOEL was not established. The confidence rating in this oral RfD is low because the study used only one dose level (therefore no dose-response relationship can be established), no NOEL was established and there was a failure to perform complete histopathological workups; these shortcomings are seen as major deficiencies of this study. Further, it is also significant to note that during the course of these experiments, an epidemic of virulent pneumonia killed a sizeable number of the rats before it was controlled. Although the survival curves were adjusted for that time, the impact of this epidemic upon the surviving animals is not assessed and would greatly call into question the utilization of this study for reliable estimation of reference dose.

Utilizing the same studies and adjusting for molecular weight, U.S. EPA has developed an ADI of 29.3 µg/day and 30.9 µg/day for antimony trioxide and antimony tetroxide respectively (U.S. EPA 1985b).

In Canada, the most recent guidelines for drinking water quality (Health and Welfare Canada, 1980) do not recommend a maximum acceptable antimony concentration in drinking water because of an insufficient toxicity data base.

The occupational threshold limit value (TLV) for antimony and its compounds is 500 µg/m^3. This value was calculated indirectly from the irritating effects of HCl because of the lack of adequate human exposure studies to antimony; it includes a safety factor of 10.

2.3 HUMAN EXPOSURE ASSESSMENT

2.3.1 Estimated Intake from Individual Sources

Inhalation and ingestion of ambient concentrations of antimony in air, food, drink and urban dust are the primary routes of exposure to humans. The predominant exposure, as with a number of other metals is by food ingestion. The following values for antimony intake are given by Reference Man:

average human ingestion (food and fluids)	10-30 µg Sb/day
average human inhalation	0.002-1.2 µg Sb/day

2.3.1.1 Soil and Dust

The distribution of antimony in the soil sampled around the Eldorado Resources Ltd. plant in Port Hope in 1986 and 1987 shows a wide range of values and is skewed largely to low concentrations. The majority of samples had antimony concentrations in the 1-10 µg/g level (37%) and in the 10-100 µg/g level (37%). Two sites had

very high antimony concentrations. The first, site #49, which is adjacent to the west bank of the Ganaraska River, had the highest concentration, 1,300 µg/g. The second-highest concentration, 340 µg/g, was found at site #2, which is located near the lakeshore bluff at the south end of Hope Street. All other values are below 60 µg/g. The mean soil concentration from all samples is calculated. With the two highest samples excluded, the mean concentration drops to approximately a third of this value. From the 1986 and 1987 (0-5 cm) data the following values were determined:

- average concentration: 53.5 µg/g
- average concentration: (excluding sites #2 and #49) 14.1 µg/g
- minimum concentration: 0.02 µg/g
- maximum concentration: 1,300 µg/g

From these concentrations, the estimated antimony intakes from soil/dust are calculated as in Table 6. The calculation of the absorbed intake is based on an assumed bioavailability of 10% for antimony in soil.

TABLE 6 ESTIMATED INTAKES OF ANTIMONY FROM SOIL/DUST INGESTION

	Child	Adult
Soil Concentrations Range Average	0.02 µg/g-1,300 µg/g 53.5 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	4.3 µg/day	1.3 µg/day
Absorbed Intake	0.22 µg/day	0.07 µg/day
Minimum Intake (Absorbed)	0.0016 µg/day (0.00016)	0.0005 µg/day (0.00005)
Maximum Intake (Absorbed)	104 µg/day (10.4)	32.5 µg/day (1.6)

2.3.1.2 Air

No area-specific air monitoring information is available. Urban levels in London, Ontario, from one high-volume sampler location (1987-1989) exhibited a mean of 0.001 µg/m^3 with a maximum of 0.01 µg/m^3 (P. Kiely, pers. comm). Annual mean air levels in the Windsor area have been reported at 0.009 µg/m^3. (IJC, 1990). A level of 0.01 µg/m^3 is assumed for this assessment.

2.3.1.3 Drinking Water

The measured levels in treated tap water from the Oshawa treatment plant for 1988 range from 0.21 to 0.77 µg/L, with a mean value of approximately 0.46. This value is assumed and intakes from drinking water are calculated as shown in Table 7.

2.3.1.4 Food

There is a lack of information regarding the dietary intakes of antimony in Ontario and Canada. The sparse information which is available span a wide range of values. For instance, a Swedish study found an average daily intake of 10 µg (Wester, 1974), while a daily intake of 34 +/- 27 µg was estimated in England. Estimates of dietary intake for man before the mid-1970s' tend to be higher than more recent values. For instance Schroeder (1970) has suggested less than 100 µg/day for U.S. diets, whereas intakes as high as 250-1,280 µg/day have been estimated in a study of institutionalized children. Using U.S. Food and Drug Administration surveys of trace metals in food, Tanner and Friedman (1977) as cited (U.S. EPA, 1980) calculate the daily intake of antimony to be too negligible to be assigned a meaningful number. Median levels were less than 0.0008 µg/g, wet weight, for most food groups. Thus there appears to be insufficient information from North Americans studies on which to suggest a dietary intake value for this substance. The FDA survey suggests that this exposure is not large.

2.4 RISK CHARACTERIZATION

The multimedia analysis of the exposure picture for antimony is impeded by the lack of reliable information from which to calculate dietary intakes for adults and children. It is thought that, in general, diet will be the predominant route of exposure, but a discrete value cannot be assigned. As a result total daily intakes and the relative contributions of exposure pathways are not calculated explicitly. Intakes through drinking water and inhalation are estimated to be very small.

The average absorbed intake calculated from soil/dust ingestion are 0.22 µg/day and 0.07 µg/day for children and adults respectively. These values are only a small fraction of any reported values for dietary exposures.

The maximum predicted absorbed intake with the highest measured concentration is 10.4 µg/day, which associated with site #49, located adjacent to the west bank of the Ganaraska River. This is a public recreational area used for fishing. Since no adjustment was made for residence time, intakes will usually be overestimated as people are likely to spend only a small portion of their time there. To account for this and provide a more plausible determination of exposure, it is reasonable to make a specific adjustment for time. For example, it can be assumed that visitors

spend 6 hours a day, 5 days a week in the area in summer, and soil is not ingested in winter if it is frozen or covered with snow or ice. Therefore direct exposure to the soil will occur only roughly 8 months a year; thus a factor of 6 hours/24 hours x 5 days/7 days x 8 months/12 months =~.1, can be applied to the soil ingested value, representing the fraction of long-term average daily ingestion of soil from the contaminated source. This would yield intakes of roughly 1 µg/day (0.06 µg/kg/day) for children and 0.2 µg/day (0.003 µg/kg/day) for adults.

TABLE 7 ESTIMATED DAILY INTAKE OF ANTIMONY: INTEGRATION OF EXPOSURE PATHWAY

Substrate or Medium	Child (1-6 years old)	Adult
SOIL/DUST		
Average Soil Concentration	53.5 µg/g	53.5 µg/g
Amount of Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake from Soil	4.3 µg/day	1.3 µg/day
Absorbed Intake	0.22 µg/day	0.07 µg/day
AIR		
Average Air Concentration	0.001 µg/m^3	0.001 µg/m^3
Volume Inhaled	5 m^3/day	22 m^3/day
Estimated Intake from Air	0.005 µg/day	0.022 µg/day
DRINKING WATER		
Assumed Tap Water Concentration	0.46 µg/L	0.46 µg/L
Daily Water Consumption	0.6 L/day	1.5 L/day
Estimated Intake from Drinking Water	0.28 µg/day	0.69 µg/day
FOOD		
Total Intake from Food	ND[1]	ND
ESTIMATED TOTAL DAILY INTAKE	ND	ND

[1] Not determinable

Besides lack of sound information from which to assess background exposures, the assessment of risk for antimony is further complicated by the lack of sound chronic toxicological data from which to derive an oral reference dose. The only oral dose located

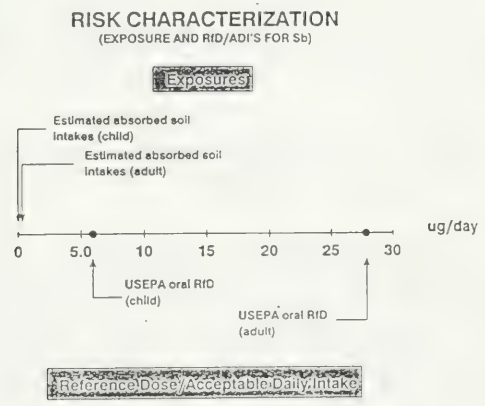
for this substance (U.S. EPA, 1991) is ranked with low confidence and therefore direct comparison to this reference dose is not strongly meaningful in predicting risk. Although the average soil intake estimates, as well as the exposure associated with the river bank are below this measure, a precise conclusion based on quantitative methods cannot be reached. Qualitatively, it would not appear, for the following reasons, that exposures to antimony levels in soils would present a health risk to those in this community:

1) There is a large margin between estimated intakes and the suggested lowest observed adverse dose in experimental animals.

2) The highest estimated absorbed intake from soil approximates the lowest reported daily dietary intake. The average intakes for adults and children are likely to be a fraction of normal daily dietary intake.

3) Antimony is thought to behave biologically and chemically in a similar manner to arsenic but to be considerably less toxic, owing primarily to a much lower solubility and bioavailability.

FIGURE 3:



3 URANIUM

3.1 HAZARD IDENTIFICATION

Uranium is a metal which exists naturally as a mixture of three radionuclides: ^{234}uranium (^{234}U), ^{235}uranium (^{235}U) and ^{238}uranium (^{238}U). Since these radionuclides undergo alpha decay, the potential exists for radiobiological effects; however, the available data suggest that in most cases chemical toxicity may be assumed to be the limiting factor with respect to human health. It is generally thought that if these were controlled, the harmful effects from radiation would be negligible. Radiological risks in relation to ingestion of measured levels of ^{238}U in soil are considered in the second part of this section in order to provide for a complete analysis.

3.1.1 Absorption & Metabolism

The metabolism of uranium as an element is very limited. Following absorption into most biological systems, uranium is generally converted to the water-soluble form found as uranyl ion UO_2^{-2}. Absorption, distribution and excretion are dependent on the form and solubility of the uranium compound as well as on the species (Wrenn et al., 1985; Berlin and Rudell, 1979). Routes of exposure to uranium include oral ingestion, inhalation and dermal absorption. For inhalation, the soluble inhaled uranium particles deposited in the upper respiratory tract are absorbed to some degree, depending on clearance mechanisms and the solubility of the compound, but most of these compounds are completely absorbed within days if they reach the alveoli. Less soluble compounds are retained in the lung tissue and associated lymph nodes for weeks or years.

It is known that some water-soluble uranium compounds are readily absorbed dermally, but insufficient human data exist to quantify this mode of uptake accurately.

Uranium is poorly absorbed by the gastro-intestinal tract, and most of the uranium ingested is passed in the feces. Experimental estimates of gastro-intestinal tract absorption have ranged from 0.1% to 30%; a recent review that included four human studies suggested a "best" estimate of 1.4% at environmental levels (Wrenn et al., 1985; Hursh and Spoor, 1973). The International Commission on Radiation Protection, ICRP, (ICRP, 1979) recommends a value of 5% for water soluble compounds and 0.2% for relatively insoluble compounds.

Following absorption, uranium is distributed mainly to the lungs, kidneys and bones. The movement of the uranium and its half-life in the different tissues depend on its solubility. The half-life of uranyl nitrate in bone is 150-200 days for rats. Depending on the animal tested, about 25-75% of the absorbed uranium concentrates in

the bones and about 5% in the kidneys. Human data show that about 1 µg/day of uranium is absorbed from food and water in the United States. About 10% of this is distributed to the kidneys. The half-life in the kidneys is 1-6 days for 99% of the absorbed dose and 1,500 days for the remainder. If absorbed, the hexavalent form is rapidly excreted by the kidney, with an overall elimination half-life of between 180 and 360 days (under conditions of normal dietary intake). The percentage uranium absorbed increases with decreasing levels of intake, with iron-deficiency, with fasting and in newborns. Individuals in these groups will have higher levels of uptake than the average person at the same level of exposure.

The following values for uranium balance are given by Reference Man (ICRP, 1984):

Intake

Food and fluids	1.9 µg/day
Airborne	7×10^{-3} µg/day

Losses

Urine	0.05-0.5 µg/day
Feces	1.4-1.8 µg/day
Others	0.02 µg/day (hair)

3.1.2 Toxicology

Uranium is considered a non-essential element with no observed bodily metabolic function.

The toxicity of uranium depends, in part, on its solubility with soluble forms being more toxic. Examples of insoluble forms are uranium metal, uranium dioxide, uranium trioxide, triuranium octaoxide and uranium tetrafluoride. Examples of soluble forms are uranium hexafluoride (which decomposes in water), uranium tetrachloride, uranyl fluoride, uranyl acetate and uranyl nitrate (miscible in water).

With respect to oral exposure, acute ingestion of uranyl nitrate (1.0 g) has resulted in vomiting, diarrhea and slight albuminuria in a human volunteer. Uranium is a classical nephrotoxin, and chronic exposure may result in kidney effects. Nephritis is the primary toxic effect of uranium in both experimental animals and humans (Hursh and Spoor, 1973; U.S. EPA, 1991). One of the initial signs of morphological damage is biochemical and functional changes such as increased urinary catalases and albuminuria, which is consistent with injury to the proximal convoluted tubules (Berlin and Ridell, 1979). These effects are seen in animals when the kidney tissue level of uranium is in the neighbourhood of 2 to 3 µg/g. This level depends on the solubility of the uranium compound taken in by the animals. In humans, epidemiological studies of uranium workers have found no increased deaths due to renal disease. However, uranium mill workers have developed statistically

significant increased excretion of beta-2-micorglobulin and five amino acids. This indicates damage to the proximal renal tubules consistent with that seen in animals. Exposure for such individuals is primarily through the respiratory tract.

There are no data with respect to the teratogenicity and mutagenicity of uranium (HWC, 1987). There are no reports of genotoxic effects following oral exposure. Preliminary studies in rats have revealed that uranium may possibly interfere with reproduction.

Developmental effects have been observed in mice after administration of 6 mg uranium/kg as uranyl acetate on gestation days 6-15, they consisted of reduced weight and length, and minor skeletal abnormalities.

Chronic exposure of rats, rabbits, guinea pigs and dogs to 0.05 to 10 mg/m^3 of various uranium compounds for 7-13 months caused no signs of injury except to the kidneys.

Rats, dogs and monkeys were exposed to 5 mg $uranium/m^3$ as uranium dioxide dust for 1 to 5 years. The rats and dogs showed no damage to the lungs. After 3 years, the monkeys showed patchy hyaline fibrosis. A second study on dogs showed slight interstitial fibrosis after 18 months past exposure. Alpha radiation levels indicated that uranium had accumulated in the lungs of the dogs and monkeys. The dogs developed foci of atypical epithelial proliferation, two benign adenomas and two adenocarcinomas with local invasion. The monkeys did not develop neoplastic changes during a 6.5 year follow-up period.

Respiratory exposure for humans has been examined in several epidemiological investigations. Studies of uranium workers have not identified any definite untoward effect. One study of 18,869 white male workers showed only transient kidney damage (proteinuria) and no evidence of permanent effects or increased mortality related to the genito-urinary system. A second study of 104 deaths in men working at six uranium mills was negative except for excess deaths due to malignant disease of the "lymphatic and haematopoietic tissue other than leukaemia".

Epidemiological studies of uranium miners have found an increase in mortality due to lung cancer, but the cause cannot be isolated to uranium. Miners are concurrently exposed to tobacco smoke, radon and its decay products, silica and other dusts, and exhaust from diesel engines. Workers at uranium-processing plants had an increase in lung cancer deaths, but smoking history was not included. The lung cancer increased with exposure to uranium along with much less exposure to radon, silica and exhaust fumes. Except in the study of dogs mentioned above, it has not been possible to demonstrate that natural uranium will cause cancer in animals. Uranium is an alpha-emitting radioactive substance and by analogy

may cause cancer since other alpha emitters, such as radon, radium and plutonium, are known to cause cancer.

Health and Welfare Canada currently classifies uranium as a Category III carcinogen, possibly carcinogenic to man. The limited evidence for carcinogenicity of uranium is confined to experiments using either highly enriched uranium compounds or highly insoluble forms administered to animals by injection or inhalation. None of the oral exposure studies in animals have demonstrated increased tumour incidence in any organ.

3.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

The current Canadian and Ontario maximum concentration for uranium in drinking water is 0.1 mg/L (HWC, 1987). The derived acceptable daily intake utilized in developing this guideline is based upon a NOAEL of 1.28 mg U/kg/day for minor histopathological lesions in the kidney observed in the (only available) subchronic ingestion study of male rabbits. Dividing this NOAEL by a uncertainty factor of 500 yielded an ADI value of 2.56 µg/kg/day. This is equivalent to 38.4 µg/day for a child and 179 µg/day for an adult.

In the derivation of drinking water criteria for uranium, U.S. EPA (1985a) derived an adjusted daily intake (AADI) of 6.0-60 µg/L, or 12-120 µg/day (based upon a 70 kg adult consuming 2 L per day and 90% of ingested uranium coming from drinking water). A similar calculation by the National Academy of Sciences, NAS, (1980a) yields a value of 35 µg/L or 70 µg/day intake, but it is based upon 10% relative source contribution from drinking water. According to IRIS (U.S. EPA, 1991), the chronic oral exposure Reference Dose (revised 10/01/89) for soluble salts of uranium is given as 3 µg/kg/day, based on a 2.8 mg U/kg/day LOAEL for moderate nephrotoxicity in rabbits (30-day oral study) to which an uncertainty factor of 1,000 was applied.

In published work regarding the 1983 national workshop for radioactivity in drinking water (Wrenn et al., 1985), it was recommended that intake of uranium in drinking water be limited by considerations of toxicity to the kidney at 187 µg/day for adults. This recommendation was based on a modified Hursh and Spoor metabolic model, a reference individual intake of 1.7 L day and an uncertainty factor of 50.

The American Conference of Governmental Industrial Hygienists (ACGIH) and the ICRP recommend that occupational exposure to uranium not exceed on average 200 µg uranium/m^3 in air. This value is derived from the tissue concentration of 3 µg uranium/mg wet weight in kidneys at which no renal damage occurs. In addition, occupational health studies of workers handling uranium at this level have shown no effects on the kidneys or blood.

3.3 HUMAN EXPOSURE ASSESSMENT

3.3.1 Estimated Intake from Individual Sources

3.3.1.1 Soil and Dust

From the 1986-1987 data on uranium concentrations in the 0-5 cm layer the following values were determined:

- average concentration (1986) 16 µg/g (n=36)
- average concentration (1987) 32 µg/g (n=41)
- minimum concentration 0.7 µg/g
- maximum concentration 135 µg/g

The average concentration for 1987 is utilized as this value is two-fold greater than the 1986 average and the 1987 survey was extended to better capture areas of public use. The average value was determined on the basis of 0-5 cm sample depths only; however the values adequately reflect the range of uranium concentrations in samples measured for 1986 and 1987.

Utilizing the above intake assumptions and concentration data, estimated daily intakes of uranium from soil/dust ingestion can be estimated (see Table 8). The estimated average intake for children is 2.6 µg/day and for adults is 0.65 µg/day. Maximum potential intakes based on the maximum concentration sample points is 10.8 µg/day for children and 2.7 µg/day for adults.

3.3.1.2 Air

The air concentration is the mean of yearly average data for 1987 and 1988 measured from three hi-volume samplers located in the vicinity of Eldorado Nuclear Ltd. The data were obtained through the Peterborough District Office.

3.3.1.3 Drinking Water

Drinking water data specific to the Port Hope area for 1981 reported levels of 0.4 to 2.6 µg/L of uranium in drinking water (HWC, 1981). Treated water data from samples taken at the Oshawa water treatment plant in 1987 averaged 0.4 µg/L. This concentration is utilized to assess exposure (although more recent information may/may not be available).

3.3.1.4 Food

Uranium is present in trace amounts in foods, and in general this accounts for the majority (approximately 98%) of a typical daily exposure for individuals. There are no data available for daily uranium intake from food Ontario or Canada; however, such values have been reported in other jurisdictions. In the U.S, studies of three urban areas suggested intake an of approximately 1.3 µg/day

TABLE 8 ESTIMATED INTAKES OF URANIUM FROM SOIL/DUST INGESTION

	Child (1-6 years old)	Adult
Soil Concentrations Average Range	32.1 µg/g 0.7 µg/g-135 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	2.6 µg/day	0.65 µg/day
Minimum Intake	0.056 µg/day	0.014 µg/day
Maximum Intake	10.8 µg/day	2.7 µg/day

(Welford and Baird, 1967). Wrenn et al. (1985) estimate daily food intake of uranium for Reference Man at 1.75 µg/day. Use of this figure also assumes no consumption of food grown in these soils.

3.4 RISK CHARACTERIZATION

Information on exposures to uranium in Ontario and Canada is limited. Health and Welfare Canada (HWC, 1987) has suggested that reasonable estimates of daily intake of uranium by adults in Canada are: air 0.002 µg; water 0.075 µg; food 1.5 µg. It was estimated that average total daily intake is about 1.6 µg, of which 99.9% is derived through ingestion. These figures do not account for background soil/dust ingestion.

For young children who are exposed to and ingest soil, the above estimates suggest that approximately 55% of uranium exposure would be from the oral ingestion of these soils/dust. For adults the contribution of the soil pathway is predicted to be less, approximately 19%. General food consumption is estimated to account for about 40% of a child's exposure and 50% of an adult's exposure to uranium in this scenario. If one considers food, water and air exposures in Port Hope as background exposures (child - 2.1 µg/day, adult - 2.7 µg/day) average soil/dust intakes would increase exposures by children by approximately 123% and adult exposure by 25%.

Comparison of both discrete soil intake estimates and total daily intakes with current acceptable daily intakes or reference doses for chronic oral exposure indicates that the modelled exposures for both adults and children to uranium in this case are well below the most conservative limits based on chemical toxicity. This is illustrated in Figure 4. For example, the estimated total

TABLE 9 ESTIMATED DAILY INTAKE OF URANIUM: INTEGRATION OF EXPOSURE PATHWAYS

Substrate or Medium	Child (1–6 years old)	Adult
SOIL/DUST Average Soil Concentration	32.1 µg/g	32.1 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake from Soil	2.6 µg/day	0.65 µg/day
AIR Average Air Concentration	0.014 µg/m^3	0.014 µg/m^3
Volume Inhaled	5 m^3	22 m^3/day
Estimated Inhaled Intake	0.07 µg/day	0.30 µg/day
DRINKING WATER Assumed Tap Water Concentration	0.418 µg/L	0.418 µg/L
Daily Water Consumption	0.6 L	1.5 L
Estimated Intake	0.25 µg/L	0.63 µg/L
FOOD Total Intake	1.75 µg/day	1.75 µg/day
ESTIMATED TOTAL DAILY INTAKE	4.7 µg/day	3.3 µg/day

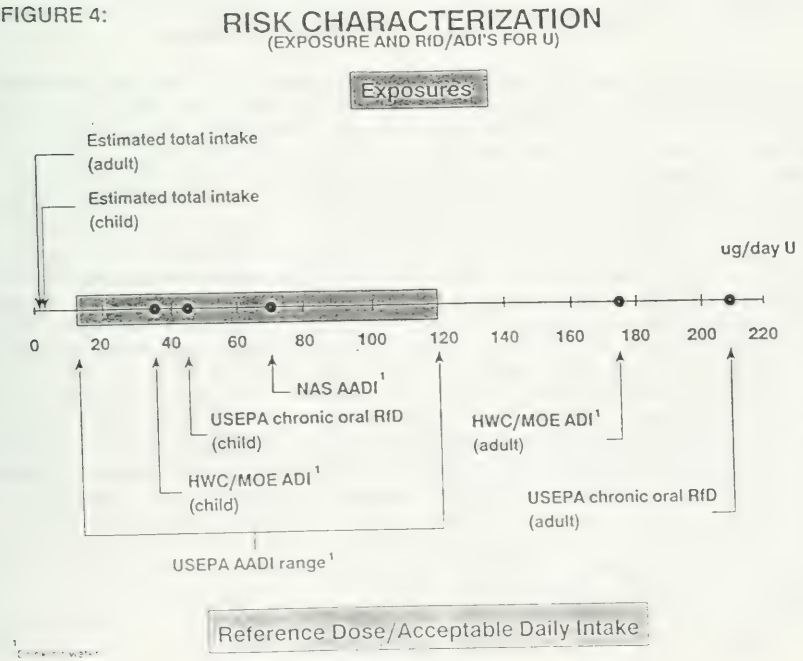
TABLE 10 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate or Medium	Percentage of Total	
	Child (1–6 Years old)	Adult
Soil/Dust	56	19
Air	2	9
Drinking Water	5	19
Food	37	53
TOTAL	100%	100%

exposure (2.6 µg/day) is less than one-tenth of the oral HWC ADI (38.4 µg/day for a 15 kg child based on ADI of 0.00256 mg/kg/day). This is also predicted for the worst case, where a soil intake of approximately 10.8 µg/day (one-fourth the ADI) for children is estimated at the highest soil concentration. This drinking water ADI contains within its derivation a safety factor of 500 applied to the no-observed-adverse effect level (NOAEL) for renal effects in rabbits. There is, therefore, a 2,000-fold difference between the highest soil intake (not the highest absorbed dose) and the effects seen in animals studies.

It is therefore concluded that exposure to uranium in the soils in question would not pose an untoward health risk to people in these areas.

FIGURE 4:



^{238}Uranium (^{238}U) Radioactivity

Although chemical toxicity is the limiting effect from environmental exposures to uranium, a discussion of the radiobiological effects and a short assessment of intakes by soil/dust ingestion is in order. Uranium is radioactive and emits particles and waves that can damage or kill cells. Uranium emits a low level of alpha particles and a much lower level of gamma rays. Alpha particles are unable to penetrate the skin but can travel short distances in the body if the element is inhaled or ingested.

Natural uranium consists of about 99% ^{238}U, and 1% ^{234}U and ^{235}U. In natural uranium the radioactivity from ^{238}U accounts for about half the total radioactivity, and the radiation from ^{234}U and ^{235}U for the other half.

Gamma rays can penetrate the skin and pass into the body. However, natural uranium releases very small amounts of gamma rays, so there is little, if any, danger from this type of radiation from uranium.

When uranium emits alpha particles, it disintegrates into other radionuclides such as thorium, radium, and radon.

The number of disintegrations per second is known as a curie (Ci). A picocurie is 10^{-12} of a curie and is equal to 0.037 disintegrations per second, or 2.2 per minute. Another unit for the same measurement is the Becquerel (Bq). One Bq = 27 pCi.
One microgram of natural uranium = 0.72 pCi. For comparison, 1 gram of radium = 1 curie, so that radium is one million times more radioactive than natural uranium.

The level of ^{238}U found in the soil at Port Hope averages 210 mBq/g of soil. If the average intake of soil for a child is 80 mg/day, then the child's intake of uranium from uranium would be 210 mBq x 0.08 g/day or 16.8 mBq/day. The occupational ALI for ingestion of ^{238}U is 400,000 Bq. This amounts to just over 1,000,000 mBq/day. The U.S. EPA recommends that a general population exposure be one-tenth of this, or about 100,000 mBq/day, a level about 7,000 times higher than the intake estimated for the child. Therefore the ^{238}U intake for a child corresponds to about 0.002% of the annual limits of intake (ALI) and 0.02% of the population exposure limit.

4 LEAD

4.1 HAZARD IDENTIFICATION

4.1.2 Absorption and Metabolism

Absorption of lead from the gastro-intestinal tract may vary depending on the age and nutrient status of exposed individuals and the matrix (e.g. food or soil) that is ingested. The rate of absortion for adult humans eating typical diets is approximately 10-15% of the ingested amount. For infants and children, the rate is generally thought to be in the order of 42-53%. This indicates a higher relative exposure rate for a given intake for children than for adults.

With respect to absorption from soil/dusts, animal and in vitro studies have demonstrated that lead in various chemical forms is as available for absorption as food lead and that the acidity of the human stomach can readily solubilize lead assimilated from soil and dust. It has been estimated that absorption of lead from soil/dusts in a child is 30%, allowing for the fact that ingestion of these materials would occur other than at meal times.

Essentially all of the lead particulate which is deposited through inhalation is absorbed, although children may experience higher deposition rates than adults.

Once absorbed, lead enters a rapid-turnover biological pool with distribution to blood, liver, spleen and kidney, or it moves into bone tissue where it is retained. Lead accumulates in body tissue over time, particularly in the renal cortex or bone. The biological half-life of lead ranges from days to years depending on the body lead pool. Excretion of lead is primarily through the urine, but it and can also be excreted in the feces (if unabsorbed) and through biliary clearance.

4.1.3 Toxicology

There is a vast database of information which documents the types and magnitude of adverse effects of lead on different tissues and organ systems, particularly in developing children. The toxicology and associated health effects from exposure to lead have been well characterized and extensively reviewed (RSC, 1986; ASTDR 1989; U.S. EPA-EEC, 1989). The following is a very brief sketch of some of the more significant types of effects in humans which have been associated with exposure to lead. For more detailed discussion the reader is encouraged to refer to the review material cited.

Lead has no known nutrient value and for that reason is a non-essential element.

It is well established that lead can affect multiple organ systems in humans. Among effects that can been seen are changes in the haematopoietic (blood-forming), renal, reproductive, cardiovascular and central nervous systems. The type and severity of the effect depends upon the level of the metal in the blood (blood lead level or PbB) and the age of the person exposed. The effects observed range from subtle molecular and cellular changes to higher degrees of functional deficit.

Much recent attention has focused upon the neurotoxic effects of lead from low-level exposure in the fetus and young child. Observations from a number of longitudinal epidemiological studies of children have provided evidence for a correlation between low-level lead exposure during early development and later neuro-behavioural performance (Mushak et al., 1990; U.S. EPA-EEC, 1989). Such impacts on neuro-behavioural performance are manifested as measured deficits in IQ, mental development indices and various elements of cognitive functions. In recent reviews of the available studies, it is suggested that the overall pattern of findings support such relationships, although no study taken alone can provide definitive evidence.

Other studies have suggested that lead may have effects on children's growth and development. Davis and Svendsgaard (1987) have concluded that the weight of available evidence suggests that exposure to low levels of lead can affect duration of gestation, birth weight and possibly other aspects of fetal growth.

Goyer (1986) has described the effects of lead on the kidney. In cases of acute exposure in children, reversible tubular function is seen. Irreversible chronic interstitial nephropathy (kidney disease) can occur at higher levels of exposure and higher blood lead levels.

The haematological effects of lead arise from perturbations of early and final steps in the biochemical pathway responsible for the synthesis of heme, an important co-factor for the physiological function of red blood cells. Interference with enzymes involved in heme synthesis can result in the accumulation of protoporphyrin in the blood. At high levels of exposure, anaemia may develop. These hematological effects of lead are reversible.

It has been suggested that exposure to lead is positively correlated with elevated blood pressure in adult males. More recent reports from well-controlled epidemiological studies support this hypothesis and provide evidence that such effects occur at much lower doses than previously suspected. It has been suggested that this effect may have no threshold (Levin, 1989). There have also been recent studies with negative findings on this correlation.

There are both positive and negative findings regarding the possible mutagenic effects of lead. Lead has been demonstrated to

affect molecular processes associated with gene regulatory mechanisms. Observations of the effects of lead with respect to sister chromatid exchange (SCE) and chromosomal aberrations have been negative in some cases and positive in others. Such effects have not been observed in children exposed to high environmental lead levels (Dalpra et al., 1983). Taken together the evidence is conflicting, but it suggests that lead may be mutagenic **in vivo**.

The carcinogenicity of lead in humans has been investigated in several epidemiological studies of occupationally exposed workers. These studies, however, provide insufficient evidence of carcinogenicity because of the presence of confounding variables, such as smoking and the presence of carcinogenic metals like arsenic. The International Agency for Research on Cancer (IARC) currently considers the overall evidence for carcinogenicity of lead to humans as inadequate (IARC, 1987). Lead and inorganic lead compounds are therefore considered as group 2B (inadequate data in humans, sufficient evidence in animals).

The carcinogenic potential of lead salts, primarily lead acetate and lead phosphate, has been investigated in more than 10 animal studies and later neurobehavioural performance. The most consistent cancer response observed is bilateral renal carcinoma. These results are considered sufficient evidence for carcinogenicity for these soluble salts by the IARC (1987) and the U.S. EPA (1989). Metallic lead and oxides have not been tested adequately. Because of its conclusion of sufficient animal evidence, U.S. EPA has classified lead in the B2 group, i.e. probable human carcinogen (human evidence is inadequate). The Carcinogen Assessment Group has recommended that numerical estimates of these risks not be utilized, as standard procedures would not truly describe the potential risk (U.S. EPA, 1991). It is also important to note that most of the animal studies indicate a carcinogenic response only at the highest dose given. It would appear that much higher doses are required for renal tumour production than for the onset of other non-carcinogenic effects. For that reason the analysis of risk from lead exposure should be based on the significant non-carcinogenic effects.

4.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

Through medical observation and scientific research, a great deal of information is available which examines the relationship between blood lead levels and various health-related outcomes. The U.S. EPA has indicated that a blood lead level of 10-15 µg/dL and possibly lower remains the level of concern for impaired neurobehavioural development in infants and young children. (U.S. EPA, 1989a). The agency considers lead to be essentially without a threshold since changes in blood enzyme, and in certain aspects of children's neurobehavioural development, may occur at very low blood lead levels. Therefore, the agency has not determined a "safe" level (RfD) for lead.

Other agencies have established intake limits for lead. The World Health Organization (WHO) has selected a provisional tolerable weekly intake of 25 µg/kg/bw, or 3.5 µg/kg/bw per day, for infants or small children. This is based on metabolic considerations and incorporate a small safety factor of 1 to 2. This is equivalent to 52.5 µg/day for a 15 kg child which is considered to be the average weight for a year old child, the most sensitive age group for lead exposure and toxic effects.

In the proposed National Primary Drinking Water for Lead (U.S. EPA, 1988a), 10-15 µg/dL in children is recommended as the appropriate range of concern for health effects that should be avoided.

In recent revising of the Canadian Drinking Water Guideline for lead, it was concluded that subtle effects which have been observed at blood lead levels in the range of 15 to 20 µg/dL, may be related to more serious clinical effects observed at higher exposure. These subtle effects include haematological perturbations, effects on vitamin D metabolism, small dose-related decrements in IQ and behaviourial tasks, and decrements in neuro-behavioural function tests. Blood lead levels of 15 µg/dL for infants and young children were considered the lowest observed adverse effect level (LOAEL). This would represent a maximum not to be exceeded for most of the population. The acceptable blood lead level was calculated using an uncertainty factor of 2 was applied to the LOAEL which results in an acceptable average blood lead level of 7.5 µg/dL in a two year-old child. The tolerable daily intake from all exposures was calculated using a blood lead:intake relationship of 0.16 µg/dL per µg/day intake, which was derived from the infant dietary study by Ryu et al. (1983). Thus the tolerable daily intake equivalent of 7.5 µg/dL PbB is 3.45 µg/kg bw per day for a 13.6 kg child.

4.3 HUMAN EXPOSURE ASSESSMENT

4.3.1 Estimated Intakes from Individual Sources

4.3.1.1 Soil and Dust

From the 1986-1987 data on lead contamination in the 0-5 cm layer in soil the following values were determined:

- average concentration 140 µg/g
- minimum concentration 1 µg/g
- maximum concentration 1300 µg/g

The confidence in individual site values and the SYMAP projections for the proposed risk assessment is diminished by the report of substantial variation between samples replicated at the same site. That is, there is uncertainty as to whether these concentrations are representative of the actual concentrations to which people would be exposed. The maximum concentration (at site #12) is

selected as 1,300 µg/g. Although there is a higher level reported for the 5-10 cm depth for this site, the very poor sample replication described in the Phytotoxicology report would exclude this as a reliable determination for exposure assessment. It is also important to note that, with few exceptions, samples taken from residential street areas are less than 200 µg/g, with many values less than 100 µg/g soil lead. Levels of 200-300 µg/g are not unusual in urban centres in Ontario and Canada. The two samples as taken in the park in children's play areas with potential for higher exposure in 1987 were 29 µg/g and 1 µg/g.

The lead intake values associated with the average and range of measured soil levels for adults and children, based on year-round daily exposure, are given in Table 11.

TABLE 11 ESTIMATED INTAKES OF LEAD FROM SOIL/DUST

	Child (1-6 years old)	Adult
Soil Concentration Average Range	140 µg/g 1-1300 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	11.2 µg/day	2.8 µg/day
Minimum Intake	0.08 µg/day	0.02 µg/day
Maximum Intake	104 µg/day	26 µg/day

4.3.1.2 Air

No specific air monitoring information for lead in Port Hope was available. For other Ontario urban centres, annual geometric means of lead are generally 0.1 µg/m^3 or less for the late 1980s' (MOE, 1989a). Therefore, 0.1 µg/m^3 will be chosen for calculating surrogate inhalation intakes of lead.

4.3.1.3 Drinking Water

The average lead concentration in treated tap water samples from the Oshawa water treatment plant in 1988 were all below the detection limit of 3 µg/L. Standing samples at two sites averaged 5 µg/L. This compares well to the value of 4.8 µg/L found in a recent Ontario study using composite sampling of lead in drinking water consumed at the tap (MOE, unpublished data). Therefore 5 µg/L is utilized as the average exposure concentration in this assessment.

4.3.1.4 Food

Food contains lead obtained from naturally occurring lead in soil as well as lead from the atmosphere, in water used for cooling and in the seams of soldered cans. Previous estimated intakes for lead in Canadian and Ontario food have suggested values of 37.4 µg/day for preschool children and approximately 63 µg/day for adult exposures based on data collected in the early 1980s' (MOE, 1991a; HWC, 1988). These values are expected to decline over the next decade (and may already have begun to decline) with the reduction of lead in air (from the phase-out of lead in gasoline), reduced lead in water and in cans. A more recent unpublished study of children in two Canadian cities suggests lead intakes of 14.8-16.4 µg/day or approximately 1 µg/kg/day (J. Salminen, pers. communication). Therefore the estimates of dietary intake for lead range from 15-38 µg/day for children in Ontario and are applied here.

4.4 RISK CHARACTERIZATION

Because of their higher contact rates with soil and higher rates of intestinal absorption for lead as compared with adults, young children will generally have greater exposures by this pathway. Although exposures of women of child-bearing age (as a surrogate for fetal exposure) merit consideration, such exposures will generally be smaller and result in smaller absorbed intakes than for children. Therefore, young children may be considered the most susceptible receptor for exposures for direct soil/dust ingestion, and therefore characterization of risk will focus on this subgroup.

It is appropriate here to discuss briefly the current scientific information relating to lead in soil and blood lead level in young children. This question has been examined to some extent in a number of epidemiological investigations. Some studies have found positive correlations between soil lead and blood lead levels in children, particularly where soil lead levels exceeed 1000 ppm. The range of reported average slope factors (which attempt to describe this relationship numerically) is 0.6-6.8 µg/dL per 1000 µg/g soil lead (MOE, 1987a,b; U.S. EPA, 1986a). The study of Baltrop et al. (1975) in Derbyshire, England, concluded that soil lead contributed 0.6 (µg/dL)/(mg Pb/g) soil in a rural area where industrial point sources of lead no longer operate. Another study has demonstrated no apparent elevation in mean blood lead concentrations (compared to low exposure groups) for children in two English villages with mean soil lead levels of greater than 1000 µg/g (Baltrop and Strehlow, 1988). In a more recent review of blood lead studies in mining areas (Steele et al., 1990), with mine waste but no recent or current history of smelting, it is noted that blood leads appear in general not to be elevated despite some very high soil lead concentrations. Average blood lead levels were lower than expected when compared with studies of urban communities or communities with operating smelters.

TABLE 12 ESTIMATED DAILY INTAKE OF LEAD: INTEGRATION OF EXPOSURE PATHWAYS

Substrate or Medium	Child (1–6 years old)	Adult
SOIL/DUST		
Average Soil Concentration	140 µg/g	140 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake from Soil	11.2 µg/day	2.8 µg/day
AIR		
Average Air Concentration	0.1 $\mu g/m^3$	0.1 $\mu g/m^3$
Volume Inhaled	5 m^3	22 m^3
Estimated Inhaled Intake	0.5 µg/day	2.2 µg/day
DRINKING WATER		
Assumed Tap Water Concentration	5 µg/L	5 µg/L
Daily Water Consumption	0.6 L	1.5 L
Estimated Intake	3 µg/day	7.5 µg/day
FOOD		
Total Intake(US data)	15–38 µg/day	63 µg/day
ESTIMATED TOTAL DAILY INTAKE	29.7–52.7 µg/day	75.5 µg/day

TABLE 13 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate or Medium	Percentage of Total	
	Child (1–6 years old)	Adult
Soil/Dust	21–37	3
Air	1–2	3
Drinking Water	6–10	10
Food	50–72	84
TOTAL	100%	100%

As a result of the complexity of exposures to lead, determining the specific contribution of any particular environmental variable like soil/dust to blood lead level is extremely difficult. This difficulty is also confounded by significant other factors such as socioeconomic status and dietary exposure. For instance, the numerous variables studied in two Ontario blood lead studies (Duncan et al. 1985; GGA, 1988) were unable to account for more than 30% of the variations seen in blood levels in children. The range of observations on the relationship between soil lead and blood lead seen in various studies is a further reflection of the difficulties of determining such associations.

Before examining the estimates of lead exposures for children and adults from soil and dust ingestion, it is useful to compare the reported levels of lead in Port Hope soils with other typical Ontario levels. Bearing in mind that there is no "typical" urban residential site, one may examine Ontario urban residential sites in built-up areas that are not obviously associated with any lead-related industry (although the areas may have been influenced to some degree by other industry, vehicle exhaust deposition, etc.). Linzon (1976) reports in a survey of an Ontario downtown area, serving as control for samples collected near a lead industry, lead levels in the 0-5 cm of soil, averaging 482 µg/g with a range of 18 to 1,450 µg/g. It is therefore reasonable to suggest that urban residential areas can experience soil lead levels of a few hundred µg/g without the influence of nearby industry. It can thus be suggested that reported on average soil lead levels in the Port Hope area are no greater than levels, and in most cases less, than those expected for other urban residential sites in Ontario. By corollary, estimated exposures to the reported levels would be crudely predicted to be on average less than that for other urban Ontario populations.

The integrated intakes for adults and children should be considered, at best, crude determinations of possible intakes as opposed to actual intakes which will vary considerably for any individual. The average estimated daily intake of lead from soil and dust ingestion together with other pathways of exposure for young children and adults are summarized in Table 12. As with most inorganic substances, dietary intake accounts for the greater contribution of daily exposures for adults and children (see Table 13). For an average adult, soil/dust exposure is estimated at 2.8 µg/day, or about 3% of daily exposure. For young children background exposure in diet will account for at least 50% and up to 72% of total exposure. The ingestion of lead from soil based on the arithmetic mean of all samples represents a relative contribution to total exposure of (between 21% and 37%, depending on the intake from diet which is chosen).

The discrete exposure from soil, if compared to the WHO provisional tolerable daily intake of 52.5 µg/day, would be approximately 20% of this value. Total combined exposure for all media (soil, air,

food and water) for children will range roughly from 30 to 53 µg/day, depending on food intake values. The values presented are averages, since actual food lead intakes values will generally vary considerably. It is most likely that the average intake from food would be towards the lower end of the range and, therefore, total intakes would likely be closer to 30 µg/day. Total average exposures for children from all pathways therefore likely represent somewhere between 60% and 100% of the WHO provisional daily intake; in light of the observation that dietary exposure in Canadian diets is declining, the 100% figure is probably an overestimate.

Measured levels and SYMAP-projected concentrations greater than 500 µg/g lead[3] are confined to two small areas: (1) the general area south of the railway viaduct on the west bank of the Ganaraska River and (2) a residential property on to Madison Street. Lead intake for a child with a daily exposure to 500 µg/g lead in soil/dust would be estimated at 40 µg/day, or about 75% of the tolerable intake. Taken together with other exposures, the total intakes would be calculated to be approximately 1.4 x the intake limit, assuming continuous daily exposure and contact with that area.

With respect to the area on the river bank, which is utilized as a fishing area by persons of all ages, this may be considered a parkland area where children will have intermittent exposure.[4] Lead exposures associated with this area will likely be some fraction of that which a continuous daily exposure estimate would suggest (e.g. 6-8 hrs/24 hrs X 104 µg/day) at the 1986 soil concentration of 1,300 µg/g. Estimated intakes from soil ingestion alone would fall below tolerable daily intakes. However, if soil concentrations in this area are several thousand µg/g (as some highly variable samples in 1986 may suggest), then exposures could exceed tolerable intakes by several fold, depending on individual contact with the soil.

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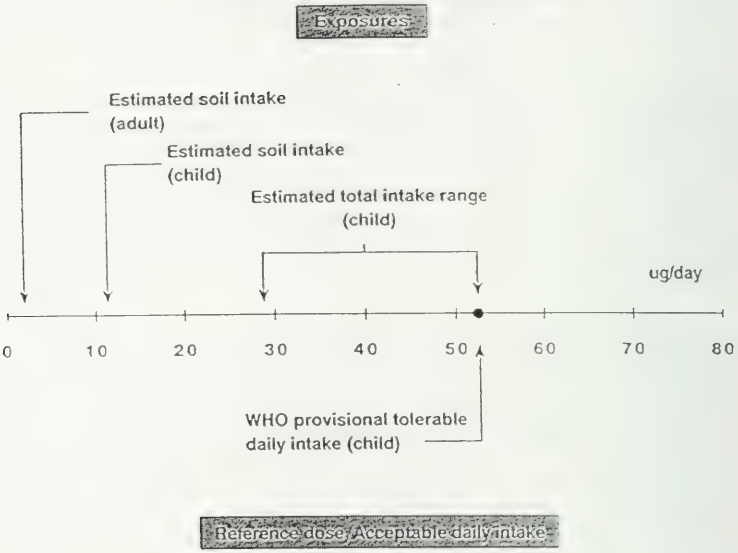
[3]In an examination of a guideline to apply to soil removal program for the lead-contaminated soil in South Riverdale area of Toronto, the lead in soil committee (MOE, 1987) concluded that "...the highest dust lead level that would keep the average young child within the maximum tolerable intake (assuming 50% comes from soil/dust) is 500µg/g." It was also concluded that this guideline was appropriate for the growing of root and leafy vegetables. It was recommended that a guideline level between 500-1,000 µg/g is appropriate for areas to which children have routine access.

[4]According to the recommendations of the Royal Society of Canada (1986) with respect to clean-up of contamination around existing plant, levels of up 10 1,000 µg/g should be acceptable for parklands and other areas where children may have intermittent access.

FIGURE 5:

RISK CHARACTERIZATION
(EXPOSURE AND RfD/ADI' S FOR Pb)



5 CHROMIUM

5.1 HAZARD IDENTIFICATION

5.1.2 Absorption and Metabolism

Chromium exists in three main valence states in nature - elemental (Cr), trivalent (CrIII) and hexavalent (CrVI). The last two are the stable forms in water.

The form of chromium affects its uptake characteristics during oral and inhalation exposures. From 0.5% to 3% of an ingested dose of inorganic salts of CrIII is absorbed in the gastro-intestinal tract whereas, 2-10% of CrVI is absorbed (Fan et al., 1987; Langard et al., 1986). The difference in the overall gastric absorption rate may reflect the ready reduction of CrVI in the gastric fluids. From 10% to 25% of organic complexes of CrIII is absorbed, and the International Commission on Radiological Protection (ICRP, 1984) states that "at least half of the chromium in food is absorbed." The chemical form of chromium in food is not known but is likely in the form of organic complexes of CrIII (Langard et al., 1986).

The studies of inhaled chromium suggest that CrVI is more readily absorbed by the lungs than CrIII, although the uptake appears to be a function of the relative solubilities of the oxychromium salts, particularly of the hexavalent chromates. Although the chromium concentrations in other tissues and organs decrease with age, that in the lung increases, suggesting that at least some of the inhaled chromium is not readily soluble.

Following absorption, chromium is distributed to blood, soft tissues - notably liver and the spleen - and bone; transport is by the iron plasma protein, transferrin. Extracellular CrVI is readily transported across cell membranes, whereas extracellular CrIII is not and undergoes intracellular reduction by the cytochrome P-450 system. Therefore, chromium apparently exists as CrIII in biological tissues, since the oxidation of CrIII is not significant.

The excretion of the absorbed inorganic CrIII, chiefly through urine (~ 80%, remainder in feces), has a quick phase (half-life of about five days) and two slower phases. The non-absorbed inorganic chromium (90-99% of the amount ingested) is excreted in the feces.

5.1.3 Toxicology

Trivalent chromium is an essential nutrient. It appears to be essential for the maintenance of normal glucose metabolism in man, although the daily requirements are not known. CrIII deficiency can lead to possible effects on growth and on lipid and protein metabolism.

Acute systemic poisoning from a single exposure to a chromium compound is uncommon. The major acute effect from ingested chromium is upon the kidneys in the form of renal tubular necrosis.

Subchronic and chronic dermal exposure to CrVI in the form of chromic acid may cause contact dermatitis and ulceration of the skin (Fan et al., 1987). Air-borne particulate CrVI salts are corrosive, causing contact dermatitis and irritation and ulceration of nasal and oral mucosa in occupational settings. The effects appear to be related to local deposition at the sites and not to the concentration in the air. Trivalent chromium compounds are considerably less toxic than the hexavalent compounds and are neither irritating nor corrosive.

There appear to be no studies of the teratogenic effects from the ingestion of chromium. Studies with rats have shown that inorganic CrIII does not cross the placental barrier to an appreciable extent (Langard et al., 1986).

There is no evidence that CrIII can act as a mutagen in *in vitro* genetic toxicological assays, because it cannot cross cell membranes. CrVI is positive in the majority of such assays because it can cross the membranes. However, it is not active in simplified systems, such as isolated nuclei or purified DNA, whereas CrIII is, since it forms tight complexes with DNA. Since CrVI is converted to CrIII inside the cells, it is CrIII that is the actual mutagen (Fan et al., 1986; IARC, 1985). Chromosomal aberrations and sister chromatid exchange (SCE) have been observed in blood lymphocytes in workers exposed to CrVI (Bianchi et al., 1985; IARC, 1985).

According to the International Agency for Research in Cancer, IARC, (IARC, 1990a), it is considered that the evidence for chromium metal and CrIII being carcinogenic in humans and animals is inadequate, and therefore they are not classifiable as to their carcinogenicity in humans (Group 3). Trivalent chromium compounds have not been reported as carcinogenic by any route of exposure. CrVI is classified as a Group 1 carcinogen (evidence considered sufficient for CrVI causing lung cancer in humans and causing cancer in animals by means of implantation and intramuscular and subcutaneous injection.) Some studies have reported cancers in humans at other sites, but the observations are not statistically significant (IARC, 1985). The Group 1 ranking for CrVI applies to the group of chemicals as a whole and not necessarily to all individual chemicals within the group. CrVI is designated by the U.S. EPA as a Class A carcinogen (human carcinogen by the inhalation route).

The evidence for carcinogenicity of CrVI is based on occupational exposures to airborne particulates in industrial processes using bichromate ($Cr_2O_7^=$) and chromate ($CrO_4^=$), such as the manufacture of pigments and alloys, the plating of metals, and the welding of stainless steel and other alloys-containing chromium (Fan et al.,

1987; IARC, 1985; Langard et al., 1986). The slightly soluble salts are more active in the lung than the highly soluble or insoluble salts, since high local concentrations of CrVI are cytotoxic and evidently mask possible carcinogenic effects. It has been suggested that the mechanism of action involves the reduction in the cells of CrVI to CrIII, which then interacts with the cell's DNA (Bianchi et al., 1985; IARC, 1985). Hexavalent chromium compounds have not produced lung tumours in animals by inhalation.

Whether chromium compounds produce cancer at sites other than the respiratory tract is not known. According to Cassaret and Doull (1986), slight increases in cancers of the gastro-intestinal tract have been reported in some occupational studies, but these involved only small cohorts of workers. There are no studies indicating that CrVI is carcinogenic when taken orally.

5.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

The current Ontario drinking water objective is 50 µg/L. The Health and Welfare Canada supporting documentation (1986) states that at the maximum acceptable concentration, "hexavalent chromium has not caused any known harmful effects on the health of man or animals. Data available are insufficient to determine whether higher concentrations are equally safe."

In 1980 the U.S. EPA set an interim maximum contaminant level for total chromium (CrIII and CrVI) in drinking water of 50 µg/L. It proposed in 1985 a maximum contaminant level goal of 120 µg/L for total chromium in drinking water. This latter value is based on a drinking water equivalent level (DWEL) of 170 µg/L, allowing for 100 µg/day from food and 0 µg/day from air. The DWEL is based on a NOAEL of 2.41 mg/kg/day in rats (one-year drinking water study done in 1958 with CrVI; uncertainty factor of 500 and 2 L/day assumed). A reference dose of 5 µg/kg/day for chronic human oral exposure to CrVI was derived from this information.

The chronic oral reference dose for CrIII is 1 mg/kg/day based on a NOEL of 1800 g/kg from a rat chronic feeding study to which an uncertainty factor of 100 was applied (U.S. EPA, 1991).

Of particular note concerning oral exposure is that an estimated adequate and safe daily dietary intake of 50 to 200 µg/day for adults has been established by the National Academy of Sciences (NAS, 1980). This range is based on the absence of signs of chromium deficiency in the major part of the United States population consuming an average of 60 µg chromium per day. This value was extrapolated to children on the basis of expected food intakes as 20-80 µg/day for one-to-two year olds and 30 to 120 µg/day for three to six year olds.

The ambient air quality criterion in Ontario is 10 µg/m^3 (24 hr), and the half-hour point of impingement value is 30 µg/m^3. In 1982,

a provisional guideline recommended an ambient air quality criterion of 1.5 $\mu g/m^3$ (24 hr) and a half-hour point of impingement value of 5 $\mu g/m^3$.

The U.S. EPA (1984) estimates that the lifetime risk from chromium in air containing 1 $\mu g/m^3$ is 1.2 x 10-2. This value is based on a 1975 epidemiological study of a chromate production facility in the United States. The cancer mortality was assumed to be due to CrVI.

5.3 HUMAN EXPOSURE ASSESSMENT

A brief description of the environmental forms of chromium is in order given the differences in toxicological properties of the valence forms. This variable toxicity of the valence form is a factor in the risk assessment of the measured soil concentrations.

Chromium exists in soil chiefly as CrIII and is found in the mineral structure or as mixed oxides of CrIII and FeII. It is only slightly mobile at pH<5.5 and is completely precipitated, complexed or adsorbed at higher pH's. CrVI is very unstable in soils and is easily mobilized at pH<6.5 and pH>8. Therefore, it will move down the soil column. It is, however, readily reduced by soil organic matter to CrIII (Fan et al., 1987).

The extractability of chromium from soils is low: <1% with most extracting agents and ~ 25% with 0.1 N HCl (Adriano, 1986). Studies have shown that both CrIII and CrVI can exist simultaneously in surface waters. CrIII is slowly oxidized in natural waters. In chlorinated drinking water, chromium is usually present as CrVI.

On the basis of the above, it will be assumed that measured chromium in soil exists almost entirely in the trivalent state for the purposes of analyzing risk from soil/dust exposures.

5.3.1 Estimated Intake from Individual Sources

5.3.1.2 Soil and Dusts

Of the 33 surface samples, only 4 were >50 $\mu g/g$. From the 1986-1987 data in the 0-5 cm layer the following values are determined:

- average concentration: 33 $\mu g/g$ (n=76)
- minimum concentration: 17 $\mu g/g$
- maximum concentration: 95 $\mu g/g$

From these concentrations, the estimated chromium intakes from soil/dusts are calculated (see Table 14). The estimated average intake for young children is 2.6 μg/day and 0.66 μg/day for adults. Maximum estimated intakes based on maximum concentrations are 7.6 μg/day for children and 1.9 μg/day for adults.

TABLE 14 ESTIMATED INTAKES OF CHROMIUM FROM SOIL/DUST INGESTION

	Child (1-6 years old)	Adult
Soil Concentrations Average Range	33 µg/g 17 µg/g-95 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	2.6 µg/day	0.66 µg/day
Minimum Intake	2.2 µg/day	0.54 µg/day
Maximum Intake	7.6 µg/day	1.9 µg/day

5.3.1.2 Air

The ambient air values for chromium in total suspended particulates in Ontario in 1987 range from 0.01 to 0.47 µg/m^3, with the majority <0.05. There are no measurements at Port Hope. An adult inhales about 20 m^3 /day. Hence, the intake from air may be estimated to be around 1 µg/day, assuming a typical concentration of 0.05 µg/m^3. The intake of a child inhaling about 5 m^3/day is around 0.25 µg/day.

5.3.1.3 Drinking Water

The concentration of chromium in measured treated tap water at Port Hope is 1-2 µg/L. An adult drinks on an average 1.5 L/day of water. The average intake of chromium is 1.5-3 µg/day. The intake of a child, who drinks about 0.6 L/day, is 0.6-1.2 µg/day.

5.3.1.4 Food

There is only meagre information on the average daily intake of Cr. The following intakes, in µg Cr/day, have been reported (Adriano, 1986):

United States:	60-280	(1977)
Japan:	130-254	(1965)
United Kingdom:	320±162	(1979)
Italy:	50	(1976)
India:	150	(1969)
Canada:	136-152	(1974)

These are presumably figures applying to adult groups.

Intake values calculated by the Hazardous Contaminants Branch from concentrations of chromium in various food groups in the Ottawa-

Hull area (Health and Welfare Canada, 1976) and from food intake data (Health and Welfare Canada, 1976; Adriano, 1986) for the same food groups are 250 µg/day for a child and 280 µg/day for an adult male. These values are assumed to apply to the populations in question.

The International Commission on Radiological Protection (ICRP, 1984) estimates an average intake of 150 µg/day for an average diet, based on available studies.

5.4 RISK CHARACTERIZATION

The multi-pathway assessment of exposure indicates that for average exposures soil intakes would represent no greater than a 1% contribution to total daily exposures. Furthermore, the calculated intake from soil is <1% of the intake from food and is about the same as that from air or water.

To characterize risk, the available exposure limits are compared with the estimated soil/dust intakes (see Figure 6).

These comparisons are made with the acknowledged uncertainty in comparing total chromium soil measurements to human reference doses based on specific valence states of the metal. For an intake of 1.5 L/day of water, the apparent "allowable" intake of hexavalent chromium, based on the Ontario drinking water objective of 50 µg/L, can be calculated to be 75 µg/day (adult) and 30 µg/day (child). Estimated intakes from soil for both age groups are very much below this value. In addition, as discussed above, it is likely that chromium in soil is largely in the form of CrIII, which is considerably less toxic than the hexavalent form.

The modelled intake from soil/dust is only a small fraction of the adult estimated adequate and safe daily intake (ESADDI) for adults and 10-15% of that for young children. Similarly, maximum calculated soil/dust intakes are also well below the lower end of the range of the ESADDI values (see Figure 6.). The values are also below the oral exposure limits contained within the U.S. EPA drinking water criteria.

The estimated soil/dusts intakes are also well below the U.S. EPA Reference Dose for trivalent Cr of 1 mg/kg/day, which is the most relevant exposure limit for soil forms of chromium.

On the basis of the above analysis, it is concluded that exposure to chromium from ingestion of soils/dust in these Port Hope soils is unlikely to result in any adverse health effects to persons in this community.

TABLE 15 ESTIMATED DAILY INTAKE FOR CHROMIUM: INTEGRATION OF EXPOSURE PATHWAYS

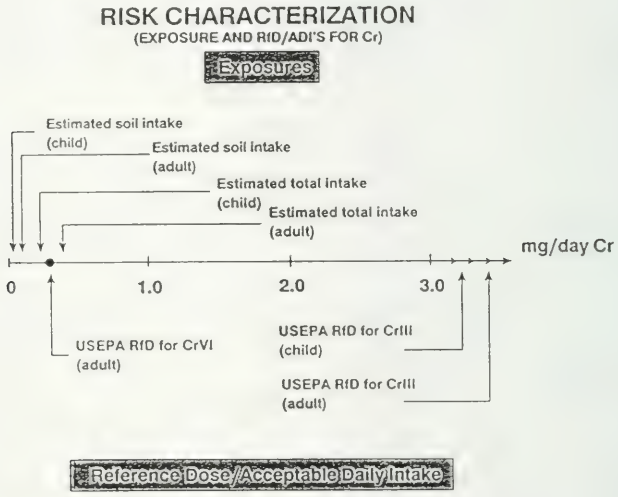
Substrate or Medium	Child (1-6 years old)	Adult
SOIL/DUST		
Average Soil Concentration	33 µg/g	33 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake from Soil	2.6 µg/day	0.66 µg/day
AIR		
Average Concentration in Air	0.05 µg/m^3	0.05 µg/m^3
Volume Inhaled	5 m^3/day	22 m^3/day
Estimated Inhaled Intake	0.2 µg/day	1.1 µg/day
DRINKING WATER		
Assumed Tap Water Concentration	1.5 µg/L	1.5 µg/L
Daily Water Consumption	0.6 L	1.5 L
Estimated Intake from Drinking Water	0.9 µg/day	2.3 µg/day
FOOD		
Total Food	250 µg/day	280 µg/day
ESTIMATED TOTAL DAILY INTAKE	254 µg/day	284 µg/day

TABLE 16 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate of Medium	Percentage of Total	
	Child(1-6 years old)	Adult
Soil/Dust	1.0	0.2
Air	0.1	0.4
Drinking Water	0.4	0.8
Food	98	99
TOTAL	100%	100%

Intakes from food generally account for almost the entire exposure for the average individual. If compared against reference doses for CrVI, the predicted dietary exposure and therefore total exposures would exceed this amount. It is likely that chromium exists as organic complexes of CrIII in food and as CrVI in chlorinated drinking water. CrVI is the biologically active species; CrIII is not readily oxidized in the body. It is therefore not possible to draw definite conclusions about the possible health effects of the suggested intake from food.

FIGURE 6:



6 COPPER

6.1 HAZARD IDENTIFICATION

6.1.1 Absorption and Metabolism

Copper is considered to be an essential element in the diet of humans, and it is widely distributed in the body. It is involved in a variety of essential bodily processes, including erythrocyte formation, the release of tissue iron and the development of bone, connective tissue and the central nervous system. In addition, copper is necessary for the proper functioning of many enzyme systems and is associated with certain elements of blood.

6.1.2 Toxicology

The biology of copper has previously been the subject of a number of more comprehensive reviews (NAS, 1977, 1980; Stokinger, 1980; WHO, 1984; U.S. EPA, 1985c) and the more salient characteristics are summarized here. Copper is considered to be an essential element in the diet of humans for proper nutrition, and it is widely distributed in the body. It is involved in a variety of essential bodily processes including erythrocyte formation, the release of tissue iron and the development of bone, connective tissue and the central nervous system. Copper is also necessary for the proper functioning of many enzyme systems and is associated with certain elements of blood.

Animal experiments indicate that deficiencies of copper are associated with anemia, depigmentation, depressed growth, bone dystrophies and gastro-intestinal disorders.

Copper is absorbed from the lungs or gastrointestinal tract following exposure. It has the highest bodily accumulations in the brain, kidney, heart liver and pancreas. Copper appears to be excreted in the feces and at a constant rate by the kidneys. Excess copper can be eliminated in feces with little change in the rate of elimination from the kidneys.

Although not thoroughly understood, homeostatic mechanisms in normal humans provide a balance between copper intake and elimination. It is known that serum copper levels may vary widely with age, sex, and hormonal and nutritional status. The homeostatic mechanisms prevent toxicity from the range of normal variations in copper.

Copper is rarely systemically toxic when ingested unless very high amounts are absorbed. Acute poisoning from oral ingestion of copper is rare owing to the powerful emetic action of this metal. However, in instances of large oral exposures, effects such as mucosal irritation, capillary damage, hepatic and renal injury and central nervous system irritation have been reported. Severe gastro-

intestinal irritation is also associated with large single doses of copper. Application of concentrated copper salts on the skin has led to papulovesicular eczema and other symptoms, reflecting the corrosiveness of the salts.

Airborne exposures to copper have occurred in the occupational setting. Pulmonary exposures have been observed to result in irritation of the respiratory tract, nausea and metal-fume fever. In some cases, discoloration of the skin and hair has been observed.

The scientific literature is replete with studies of the nutritional essentiality of copper, but copper toxicity from chronic exposure has not been well investigated. There are no data indicating that human exposure to copper results in chronic toxic effects. Diets containing up to 5.8 mg of copper/day have produced no noticeable effects in humans. Long-term inhalation of copper fumes and fine aerosols may result in metal-fume fever. Wilson's disease (hereditary hepatolenticular degeneration), a rare inborn error of metabolism, appears to be the only manifested form of chronic copper toxicity by ingestion in humans. This is a condition characterized by bodily retention of high levels of copper, accumulating in brain, liver and kidney.Another group at increased risk from chronic high level ingestion are individuals with glucose-6-phosphate deficiencies.

There is no evidence that any copper compounds are carcinogenic. The International Agency for research on Cancer has not evaluated copper or copper compounds for carcinogenicity. Copper is classified by the U.S. EPA in the Group D, inadequate data in humans and animals.

6.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

In the case of copper there are basically two types of limits to consider; 1.) Recommended daily intakes which are based on a minimum nutritional requirement and 2.) Maximum permissible intakes.

For children, a World Health Organization recommended daily intake (WHO-RDI) of 0.08 mg/kg body weight (1.2 mg/day for a 15 kg child) is thought to be necessary for proper development. The National Academy of Sciences (1980) has a recommended daily allowance (RDA) for copper of 2.0-3.0 mg/day for adults and 1.5-2.5 mg/day for children. The 1980 estimated safe and adequate daily dietary intake (ESADDI) ranges for the adult diet is 2.0-3.0 mg/day; the ESADDI ranges for the infant and toddler diets were 0.7-1.0 mg/day and 1.0-1.5 mg/day respectively.

The joint FAO/WHO Expert Committee on Food Additives has set a limit of 0.5 mg/kg body weight (7.5 mg/day for a child; 35 mg/day for an adult) as an acceptable maximum daily intake for copper.

In the development of a drinking water criterion for copper, the U.S. EPA (1985), has focused on various human studies which suggest that ingestion of between 5.3 and 32 mg of copper/person resulted in gastro-intestinal disorders, vomiting, nausea and diarrhea. Because no lasting adverse effects were reported and the symptoms have been the result of local gastro-intestinal irritation, the single oral dose of 5.3 mg was considered as a lowest observed adverse effect level (LOAEL). In determining an ADI for copper, the WHO applied a safety factor of 2 to this LOAEL, yielding a value of 2.65 mg/day. These data were also used to calculate a lifetime adjusted ADI, as the acute effects appear to be the effects of concern from exposure to copper.

6.3 HUMAN EXPOSURE ASSESSMENT

6.3.1 Estimated Intakes from Individual Sources

6.3.1.1 Soil and Dust

From the 1986-87 data on copper concentrations in the 0-5 cm layer in soil the following statistics were determined:

- average concentration 38 µg/g
- minimum concentration 3 µg/g
- maximum concentration 320 µg/g

The average was based on 0-5 cm samples only; however the values represent the range of concentrations encountered in all sample points. The maximum concentration was found at only one site. The next highest level is 225 µg/g. The estimated intakes of copper from soil and dusts for adults and children is shown in Table 17.

TABLE 17 ESTIMATED INTAKES OF COPPER VIA SOIL/DUST INGESTION

	Child (1-6 years old)	Adult
Soil Concentration Average Range	38 µg/g 3-320 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	3 µg/day	0.8 µg/day
Minimum Intake	3 µg/day	0.8 µg/day
Maximum Intake	26 µg/day	64 µg/day

6.3.1.2 Air

Results of the air monitoring program for Ontario suggest annual mean concentrations of copper in particulate of 0.017 µg/m^3 for various Ontario locations. As no air monitoring data for copper specific to the site was located this average concentration is assumed.

6.3.1.3 Drinking Water

The drinking water concentration of copper, based on treated samples at the Oshawa water treatment plant for 1986-87, averaged 4 µg/L. Data from standing samples in the distribution system range from 22-49 µg/L. Data from consumer taps in the area where soil surveys were conducted were not available. It should also be considered that consumer tap water may contain more copper than the original water supply because of the dissolution of copper from copper piping, which is common in residential housing. The most relevant data of this type comes from a integrated monitoring survey of consumed tap water in Ontario (MOE, 1989b). The average concentration of copper in water over a one week sampling period was 176 µg/L. Therefore this value is utilized as a conservative value for the drinking water intake estimate.

6.3.1.4 Food

Dietary copper is generally the primary source for copper exposure. No figures specific to Ontario were found that would provide an estimated intake for this media. Average dietary intakes as determined by the U.S. Food and Drug Administration for 1980 were assumed (Pennington et al., 1984 as cited in U.S.EPA, 1985). Use of these figure also assumes no consumption of foods grown on site.

6.4 RISK CHARACTERIZATION

The integrated exposure estimates and assumptions employed for typical adults and children are described in Table 18. This exposure model provides crude estimates of possible copper intakes from various media in this vicinity. The very limited data on water and air concentrations limit the value of these estimates. Also the value assumed for food on the basis of Food and Drug Administration studies of U.S diets, does not necessarily hold for Ontario. The estimated intakes suggest that dietary copper is by far the largest contribution to total daily intake.

The estimated intake from soil/dusts are 3 µg/day for young children and 0.8 µg/day for adults, based upon the average soil copper concentrations, and represent a relatively minor contribution to total intake (0.4% for children, negligible for

TABLE 18 ESTIMATED DAILY INTAKE OF COPPER: INTEGRATION OF EXPOSURE PATHWAYS

Substrate or Medium	Child (1-6 years old)	Adult
SOIL/DUST Average Soil Concentration	38 µg/g	38 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake from Soil	3 µg/day	0.8 µg/day
AIR Average Concentration in Air	0.17 µg/m^3	0.017 µg/m^3
Volume Inhaled	5 m^3/day	22 m^3/day
Estimated Inhaled Intake	0.1 µg/day	0.4 µg/day
DRINKING WATER Assumed Tap Water Concentration	176 µg/L	176 µg/L
Daily Water Consumption	0.6 L	1.5 L
Estimated Intake from Drinking Water	109 µg/day	264 µg/day
FOOD Total Intake	680 µg/day	1600 µg/day
ESTIMATED TOTAL DAILY INTAKE	792 µg/day	1865 µg/day

adults). The relative contribution of each pathway to total exposure is shown in Table 19.

In order to characterize the potential risks associated with copper contamination of soil, the age-specific exposure determinations are compared to current acceptable exposure limits for this substance (see Figure 7). The estimated total exposures (792 µg/day/child; 1.865 mg/day/adult), utilizing copper intake from average soil levels, fall below the NAS ESADDI ranges of 1.0-1.5 mg/day for children and 2.0-3.0 mg/day for adults, the FAO/WHO acceptable maximum daily intake of 0.5 mg/kg/day (7.5 mg/day for a 15 kg child) and the ADI value of 2.65 mg/day suggested by the 1985 U.S EPA Drinking Water Criteria document. Maximum intake based on the

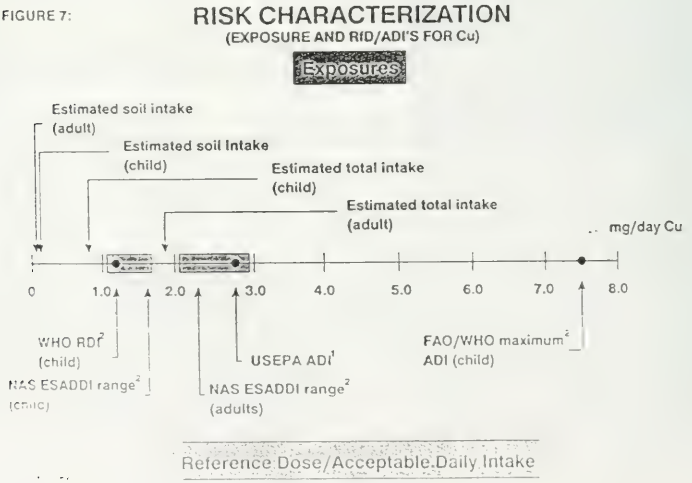
maximum soil concentration would not add appreciably to daily intake.

The copper levels in soil on these sites were estimated to result in exposures which are below currently recommended maximum acceptable health limits. These are not predicted to pose any appreciable risk to area populations based upon the small contribution of soil/dust to total exposure (0.4%, child and less than 1% for adults) and total multimedia intakes well below current exposure limits.

TABLE 19 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate or Medium	Percentage of Total	
	Child(1-6 years old)	Adult
Soil/Dust	0.4	0
Air	0	0
Drinking Water	14	14
Food	86	86
TOTAL	100%	100%

FIGURE 7:



7 NICKEL

7.1 HAZARD IDENTIFICATION

7.1.1 Absorption and Metabolism

Nickel may exist in oxidation states from -1 to +4, but the most prevalent one in the environment is +2 (NiII). The gastro-intestinal intake of nickel in food can be quite high, because of contributions from utensils and equipment used in processing and preparing food. However, the absorption of even soluble nickel compounds is only 2-5%, although it may be higher on an empty stomach. Bioavailability also appears to depend on dietary composition.

The amount of inhaled particulate nickel absorbed from the pulmonary tract is a function of both the chemical and physical form of the particles. Insoluble particulate nickel is absorbed only very slowly, whereas soluble nickel salts are cleared rapidly (in a few hours to several days). Some of the insoluble particles are cleared from the lungs and swallowed.

Absorbed nickel is carried by the blood to the other parts of the body. Albumin is the main macro-molecular carrier in man and other species. The transfer to the kidneys is quick, and the main route of excretion of absorbed nickel is urine, whereas biliary excretion is negligible in animals. Whether it occurs in humans is unknown. Unabsorbed dietary nickel is lost in the feces. Because of the low extent of gastro-intestinal absorption, fecal elimination roughly equals the daily intake. Animal studies suggest that absorbed nickel has a very short half-life in the body, in the order of several days; there is little evidence for tissue accumulation once exposure has stopped.

Age-dependent accumulation appears to occur only in the lung. This can be explained by an active phagocytic mechanism of insoluble or slightly soluble nickel compounds. Once inside the cells, the particles form an intracellular source of nickel ions that are bioavailable. The active phagocytic uptake appears to be related to the surface charge of the particles (negatively charged are phagocytized) and their size.

Transplacental transfer has been demonstrated experimentally in rodents, and there is evidence that NiII crosses the placenta in humans.

7.1.2 Toxicology

The toxicology of nickel and nickel compounds has been previously reviewed in detail (Stokinger, 1981a; MOL, 1986; ECETOC, 1989; IARC, 1990a; ASTDR, 1987). The information presented in this section is drawn from these reviews as well as being augmented by the very recent conclusions of an International Committee.

Nickel is an essential nutrient in several species and may, in minute amounts, have essential biochemical functions in humans, although this has not been established conclusively. Nickel deprivation in mammals has an adverse effect on body weight, on reproductive capability and on the viability of offspring and it induces anemia through reduced absorption of iron. Nickel also appears to be required in several proteins and enzymes (MOL, 1986).

Absorbed nickel affects the immune system. Sensitization for contact dermatitis occurs from absorption through the skin of soluble nickel compounds which are released through the corrosion by sweat of objects made of nickel metal and alloys. A large fraction (10-20%) of the female population is sensitized during the teen years as a result of daily skin contact with objects made from nickel alloys. Sensitization may also occur from occupational exposures. Provocation of dermatitis (hand eczema) occurs through additional dermal contact and is also possible after ingestion.

Respiratory allergy in the form of asthma can result from occupational exposures to aerosols from nickel plating or polishing of nickel alloys or to welding fumes.

Nickel has been shown in experimental studies to cause immunotoxic effects, such as increased infectiousness, decreased natural killer-cell activity, inhibition of interferon production and interference with functions of the complement system. Although some of these effects have been demonstrated at concentrations likely to occur under occupational exposure conditions, the health significance of these findings is difficult to evaluate. It has been speculated that, since the immune system has important functions in the defence against foreign cells, its weakening could possibly be related to the increased occurrence of respiratory cancers in occupational settings.

There is no information on reproductive effects in humans, even at occupational doses. However, experimental studies with animals have demonstrated effects at doses that are higher than the likely human exposures. Reduced fertilization rate has been found in male mice, and decreased birth weight, reduced number of live pups per dam and increased number of neonatal deaths have been observed with female mice and rats. Significant embryotoxicity and malformations were found when mice were injected intra-peritoneally.

Nickel appears not to be mutagen in bacteria or phages but is possibly a mutagen in mammalian cells. However, the experimental data are of poor quality. It may also cause DNA lesions. Experimental results from animals provide doubtful evidence for chromosomal damage in vivo. There is weak positive evidence for chromosomal damage in exposed workers. Increased rates of sister chromatid exchange (SCE) have been found in vitro but not in exposed workers.

Studies of cellular transformations in vitro are almost all positive, and epithelial changes have been found in exposed workers. Manganese appears to prevent such transformations in vitro. The transformations are enhanced if organic carcinogens are added together with the nickel. Cigarette smoke appears to have a synergistic effect.

There is no evidence that ingestion of nickel causes cancer in humans. In the single study (Schroeder et al., 1964) when nickel was administered orally in drinking water, the tumour frequency in exposed animals was less than in the controls. No tumours developed when the mucosa of the cheek pouches of hamsters were painted with nickel subsulfide (Ni_3S_2) (Sunderman et al., 1978). However, there are numerous rodent studies where nickel compounds are carcinogenic via in and that the potential varies for different compounds. The experiments suggest that the NiII ion is the ultimate carcinogen (MOL, 1986).

Inhalation of finely powdered nickel metal by rats and hamsters produced carcinogenic effects in the upper pulmonary tract, but the results were negative with nickel oxide. Epidemiological studies indicate that various forms of nickel cause cancer of the nasal cavities and lungs of exposed workers. The evidence is not clear-cut, but exposure to a mixture of oxidic and sulfidic nickel at very high concentrations causes cancer. Exposure to oxidic nickel at large concentrations in the absence of sulfidic nickel is also associated with an increased risk of lung and nasal cancer. There is evidence that soluble nickel increases the cancer risks and may enhance risks associated with exposure to less soluble forms. There is no evidence that metallic nickel causes lung or nasal cancers, and there is no substantial evidence that occupational exposures to nickel in any form is likely to cause cancer in other parts of the body. The available evidence suggests that respiratory cancer risks are primarily related to exposures to soluble nickel at concentrations >1 mg nickel/m^3 and to exposures to less soluble forms at concentrations >10 mg nickel/m^3 (ICNC, 1990).

IARC (1990) concludes that nickel compounds are carcinogenic to humans as there is sufficient evidence (nickel sulfate and the combinations of nickel sulfides and oxides encountered in the nickel refining industry) cause lung and nasal cancer. Metallic nickel is possibly carcinogenic to humans although there is only inadequate evidence in humans. The evidence in animals is sufficient for metallic nickel and nickel sulfides and limited for nickel alloys and some nickel salts.

7.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

The U.S. EPA has developed an oral reference dose for non-carcinogenic effects of 20 µg/kg/day. It is believed that a daily exposure at this level is without an appreciable risk of

deleterious effects to the human population, including sensitive subgroups, during a lifetime. The RfD is based on a NOAEL found in a study where rats were fed a diet containing 100 µg nickel/g salts (equivalent to 5 mg/kg/day). The results were decreased body and organ weights. An uncertainty factor (UF) of 100 was multiplied by a modifying factor of 3 because of certain inadequacies in the study and divided into the NOAEL to get the RfD (U.S. EPA, 1991).

Ontario has not set an objective for drinking water. In 1985 the U.S. EPA set a lifetime adjusted acceptable daily intake (AADI) of 350 µg/L for nickel in drinking water. This is equivalent to a daily intake of 700 µg/day, assuming a consumption of 2 L/day of water. The AADI is based on the same study as the 1988 RfD value of 20 µg/kg/day, which translates into an intake of 1400 µg/day for a 70 kg person. In the 1985 calculations, the ADI was divided by an uncertainty factor of 100 and multiplied by a further factor of 0.2, representing an assumed difference in absorption of nickel in water versus milk to allow for the fact that the nickel was not given to the rats in drinking water.

Ontario has set an interim air quality standard (24 hour averaging period) of 2 µg nickel/m^3, with a limiting effect based on vegetation effects and soiling.

The U.S. EPA has estimated that the incremental unit risk for lung cancer due to lifetime occupational exposure to nickel refinery dust containing 1 µg nickel/m^3 is 2.4×10^{-4}, based on epidemiological data. This value is the mid-point of the range of risk estimates from 1.1×10^{-5} to 4.6×10^{-4}, based on studies from different refineries. The quantitative unit risk estimate for Ni_3S_2, which is the most carcinogenic nickel compound in animal studies, is twice that for nickel refinery dust because it makes up about 50% of the refinery dust.

7.3 HUMAN EXPOSURE ASSESSMENT

Although actual speciation data for nickel in soil are not found in the literature, nickel may exist as minerals (chiefly ferromagnesium minerals or precipitates) and as free ion chelated metal complexes in soil solution or adsorbed on surfaces. A lowering of the pH will release nickel from the soil. As pH increases, nickel adsorption by iron and manganese oxides increases, and at pH>9, nickel carbonate or hydroxide may precipitate (Adriano, 1986).

NiII, which is the predominant form in fresh waters at pH 5 to 9, forms stable complexes in water with inorganic and organic ligands. It is also associated with iron and manganese oxides. Therefore, generally >95% of nickel in rivers and about 40% in lakes is in particulate form. About 40% of the total nickel in water is bioavailable (Moore and Ramamoorthy, 1984).

Nickel in air is in particulate form. The predominant forms appear to be nickel sulfate (from combustion of fossil fuels, the predominant source); complex oxides of nickel and other metals, chiefly iron; nickel oxide; and, in small quantities, metallic nickel and Ni_3S_2 (IARC, 1990a; U.S. EPA, 1986b).

7.3.1 Estimated Intake from Individual Sources

7.3.1.1 Soil and Dust

The sampling of soil in Port Hope around the Eldorado Resources Ltd. plant in 1986 and 1987 indicates that the distribution of total nickel in the soil is quite uniform, both regardless of distance from the plant and with depth. There are a few random elevated concentrations. Of the 36 surface samples collected in 1986 (0-5 cm), only 2 were >40 µg/g (air dried). In 1987, of the 41 samples collected, two were >40 µg/g.

- average concentration 14 µg/g (n=76)
- minimum concentration 4 µg/g
- maximum concentration 130 µg/g

Based on these concentration, the estimated nickel intakes from soil/dust are calculated and shown in Table 20.

TABLE 20 ESTIMATED INTAKES OF NICKEL FROM SOIL/DUST

	Child (1-6 years old)	Adult
Soil Concentration Average Range	14 µg/g 4 µg/g - 130 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	1.1 µg/day	0.3 µg/day
Minimum Intake	0.3 µg/day	0.08 µg/day
Maximum Intake	10.4 µg/day	2.6 µg/day

7.3.1.2 Air

The ambient air concentrations for nickel in total suspended particulates in Port Hope arer not known. Ambient air in urban areas usually contains geometric mean concentrations of less than 10 ng/m^3 (MOE, 1988). This concentration is assumed to develop the estimates of inhaled intake in Table 21.

7.3.1.3 Drinking Water

The concentration of nickel in drinking water at Port Hope is not known, but median concentrations in Lake Ontario are 0.7 to 1.6 µg/L (Rossmann and Barres, 1988). The Ministry of the Environment drinking water monitoring program found in 1988 that the concentrations in the Oshawa water distribution system ranged from less than the detection limit to 5.2 µg/L. Water that has been standing in the plumbing for several hours may reach concentrations around 15 µg/L. Based on an average concentration of 2 µg/L from the Oshawa treatment plant and an average of 1.5 L/day of water, the intake by an adult is then about 3 µg/day. The intake by a child who drinks 0.6 L/day is about 1 µg/day.

7.3.1.4 Food

There is only meagre information on the average daily intake of nickel. The following intakes, in µg nickel/day, have been reported (ICRP, 1984; MOL, 1986; IARC, 1987):

- USA: 160
- Italy: generally <300
- Denmark: 155
- USSR: 290-500

A 1974 study showed that the Canadian intake of nickel ranged from 347 to 576 µg nickel/day, with a mean intake of 460 (Adriano, 1986). This value is above the upper limit of most other countries. A value of 350 µg/day will be utilized as the dietary intake figure for adults and children.

Nickel can also be leached from stainless steel utensils, especially at low pH. Food processing, such as the milling of flour and catalytic hydrogenation of fats and oils with nickel catalysts also can add nickel. Intakes may reach as high as 1,000 µg/day.

7.3.1.5 Dermal

Dermal absorption of nickel from soil on the skin is also a possible exposure route. Dermal absorption of soluble nickel salts and from nickel metal and its alloys has been demonstrated. As the chemical form of nickel in soil is not known, it is not possible to estimate with any precision how much can be solubilized and therefore absorbed when soil is in contact with the skin. Uptake via this route is thought to be very minor.

7.4 RISK CHARACTERIZATION

The multimedia exposure assessment clearly predicts that average intakes of nickel from soil and dust represent a very small fraction of the total daily exposures. Air exposures are trivial for both child and adults, whereas diet is likely to account for

TABLE 21 ESTIMATED DAILY INTAKE OF NICKEL: INTEGRATION OF EXPOSURE PATHWAYS

Substrate or Medium	Child (1-6 years old)	Adult
SOIL/DUST		
Average Soil Concentration	15 µg/g	15 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake from Soil	1.2 µg/day	0.3 µg/day
AIR		
Average Air Concentration	10 ng/m^3	10 ng/m^3
Volume Inhaled	5 m^3	22 m^3
Estimated Inhaled Intake	0.05 µg/day	0.2 µg/day
DRINKING WATER		
Assumed Tap Water Concentration	2 µg/L	2 µg/L
Daily Water Consumption	0.6 L	1.5 L
Estimated Intake	1.2 µg/day	3 µg/day
FOOD		
Total Intake (US data)	350 µg/day	350 µg/day
ESTIMATED TOTAL DAILY INTAKE	352.5 µg/day	353.5 µg/day

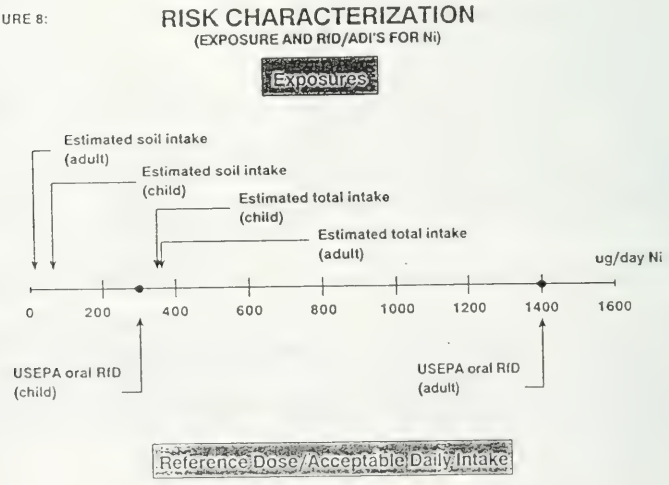
TABLE 22 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate or Medium	Percentage of Total	
	Child (1-6 years old)	Adult
Soil/Dust	0.3	0.1
Air	0	0.1
Drinking Water	0.7	0.8
Food	99	99
TOTAL	100%	100%

99% of total intake for typical individuals. All the available evidence suggests that nickel is carcinogenic only when inhaled in particulate form and at concentrations likely to be found mainly in occupational exposures. As inhalation is a trivial exposure here, the principal consideration in assessing possible health effects from nickel in soil exposure at Port Hope are threshold considerations. The U.S. EPA oral RfD of 20µg/kg/day, based on feeding studies with rats, is equivalent to a total daily intake of 1,400 µg for an adult (70 kg bw) and 300 µg for a child (15 kg bw). As the RfD was not adjusted for absorption efficiency, the intake values estimated can be compared directly with the allowable daily intake.

The estimated average intakes from soil for children and adults are .37% and 0.02% of the reference dose. The maximum intakes associated with the highest measured concentrations are approximately 3.5% of the reference dose in the child case and 0.18% in the adults case.

FIGURE 8:



The Canadian intake of nickel from food - about 350 µg/day - is roughly 30-40% of the allowable adult intake and the mean and maximum intakes from ingested soil are 0.1% and 0.6% of the food intake. It should be noted that the intake of nickel from food in other countries is about one third of the Canadian intake. The total estimated intakes are below the adult RfD.

The total intake of food by children is somewhat less than that for adults, and the components of the diet of children differs. As an initial estimate, in the absence of other data, the intake of nickel from food can also be taken as 350 µg/day in Ontario. This nickel intake from food is about 15% above the U.S. EPA reference dose for children, although this conclusion is uncertain, especially as the intakes measured in other countries are about 40% of this reference dose. Therefore the estimated total daily intake for children is slightly higher than the reference dose.

It is concluded that potential exposures from Port Hope levels reported to do not pose a health risk to persons in these areas because (1) nickel is an essential dietary component, (2) a very small percentage of total exposure is contributed by soil/dust ingestion, and (3) soil exposure is only a small percentage of the oral reference dose for humans.

8 CADMIUM

8.1 HAZARD IDENTIFICATION

8.1.1 Absorption and Metabolism

Ingested cadmium in food or water is absorbed with rather poor efficiency (1-6%) from the gastro-intestinal tract of humans and animals. This efficiency is affected by the chemical form of cadmium, dose level, age and sex. Once absorbed, it is widely distributed with preferential accumulation in kidney and liver. Excretion occurs in urine and feces but very slowly and therefore cadmium can accumulate over time in exposed organisms (ASTDR, 1987).

8.1.2 Toxicology

Cadmium may produce multiple systemic effects in both animals and humans, including injury to the kidneys, liver, cardiovascular system and immune system. Similar types of effects are observed after inhalation or oral exposure. The nature and extent of effects observed will depend upon the level of exposure. Acute ingestion resulting from relatively high concentrations of cadmium is associated with nausea, vomiting and abdominal pain. The primary health considerations have been for chronic low-level exposure by either the oral or inhalation route. The principal effects associated with such exposures include chronic renal tubular disease, emphysema and chronic obstructive lung disease. It is generally thought that the degree of severity of such effects is directly dependent on the level of cadmium in the respective target tissues. In most instances, the kidney is identified as the most sensitive tissue or critical organ. Decreased kidney resorption of low-molecular-weight proteins (proteinuria) is considered the most reliable indicator of renal injury.

Cadmium exposures have not been observed to result in reproductive, teratogenic or other developmental effects in exposed humans populations (Bernards and Lauwerys, 1984). However, in animal studies there is some evidence of teratogenicity and embryotoxic effects associated with parenteral administration of cadmium compounds; however these effects are generally not observed following oral exposure.

With respect to cancer, cadmium is considered a probable human carcinogen (category 2A) by the International Agency on Cancer Research (IARC) based upon limited evidence from epidemiological studies but sufficient evidence in rats and mice by two routes of exposure. The U.S. EPA currently classifies cadmium in the B1 group, signifying that it is a probable human carcinogen by inhalation. This agency has calculated an inhalation unit risk for excess lung cancers from human data of 1.8×10^{-3} per $\mu g/m^3$ cadmium, utilizing a two-stage extrapolation model. Several studies have

suggested an increased frequency of prostate cancer in exposed occupational groups, but this evidence is considered too weak to conclude that cadmium is a prostate carcinogen (U.S. EPA, 1985b).

Of more relevance to the question of oral exposure to cadmium is the summary report by U.S. EPA (1990) indicating that seven separate studies in rats and mice where cadmium was administered orally have shown no evidence of a carcinogenic response (U.S. EPA, 1991). There is insufficient data to classify cadmium as carcinogenic to humans by the oral ingestion route.

8.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

As indicated above, the critical organ for systemic toxicity is the kidney; the effects are related to the kidney tissue levels of cadmium. A concentration of approximately 200 µg cadmium/g wet weight of human renal cortex is considered the highest kidney level not associated with significant proteinuria (ASTDR, 1987). It has been estimated that a daily intake of 0.4-0.5 mg/day would lead to only 0.1% of the population reaching this level (Piscator, 1985). In the development of a reference dose for chronic oral exposure, U.S. EPA has employed a toxico-kinetic model to calculate the level of chronic oral exposure which would result in this tissue level. The model predicts a chronic NOAEL for cadmium of 0.005 mg/kg/day for water (assuming 5% absorption) and 0.01 mg/kg/day for food (assuming 25% absorption). An uncertainty factor of 10 was applied to account for sensitive individuals, yielding a RfD of 5×10^{-4} mg/kg/day (water) and an equivalent 1.0 µg/kg/day for food. The confidence level in these RfD values is high, because the choice of NOAEL reflects the data from numerous toxicity studies in humans and animals and the data support pharmacokinetic modelling (U.S. EPA, 1990). These values suggest a RfD range of 7.5-15 µg/day for a child and 35-70 µg/day for an adult.

A joint expert committee of the Food Additives Organization and the World Health Organization has recommended a provisional tolerable weekly intake of 0.4-0.5 mg/week, or 0.057-0.071 mg/day per individual (WHO, 1972, 1984).

8.3 HUMAN EXPOSURE ASSESSMENT

8.3.1 Estimated Intake from Individual Sources

Exposures to cadmium can occur from water, food, ambient air, occupational settings and smoking. In general food is the greatest source of cadmium exposures. Smoking can approximately double an individual's cadmium intake (ASTDR, 1987). Except in special situations, intake of cadmium from drinking water and ambient air will be of minor significance. Cadmium has not been observed to result in significant health effects when exposure is by the dermal route (ASTDR, 1987) and therefore this exposure is not quantified here.

8.3.1.1 Soil and Dust

The reported 1986 and 1987 soil survey values are considered together. The 0-5 cm sample set is selected as representing soil/dust contact concentrations. For 1986 values, the mean concentration is 0.5 µg/g, and for 1987, the mean is 0.47 µg/g. Therefore a mean of 0.5 µg/g will be utilized. For exposure estimation, the following values are utilized:

- average concentration 0.5 µg/g (n=76)
- minimum concentration 0.1 µg/g
- maximum concentration 2.1 µg/g

Estimated daily intake of cadmium from these soils are estimated as shown in Table 23. The estimated average cadmium intake for children is 0.04 µg/day and for adults is 0.01 µg/day. At the maximum measured concentration, maximum resulting intakes are 0.16 µg/day and 0.4 µg/day for children and adults respectively.

TABLE 23 ESTIMATED INTAKES OF CADMIUM FROM SOIL/DUST INGESTION

	Child (1-6 years old)	Adult
Soil Concentrations Average Range	0.5 µg/g 0.1 µg/g-2.1 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	0.04 µg/day	0.01 µg/day
Maximum Intake	0.16 µg/day	0.4 µg/day

8.3.1.2 Air

No ambient air measurements for cadmium in Port Hope are available. Ontario Ministry of the Environment statistics for cadmium in total suspended particulate in air for 1988 indicate typical arithmetic mean concentrations of 0-0.003 µg/m^3 in urban locations (MOE, 1989a). Higher concentrations may be expected in heavily industrialized areas, particularly near smelting operations.

8.3.1.3 Drinking Water

It is expected that consumption of drinking water from unpolluted sources will result in only a very small contribution to total daily intake of cadmium. It has been estimated that in the majority of cases in Canada, water contributes less than 0.01 mg/day (HWC,

1990). No drinking water data for cadmium specific to the Port Hope area was available. As a surrogate, levels in treated water samples from the Oshawa water treatment plant were used (MOE, 1989b). Except in three samples, all concentrations were below detection limit. Therefore the detection limit of 0.3 µg/L is selected for exposure calculations.

8.3.1.4 Food

The principle exposure to cadmium for most individuals is from food. Results of a duplicate diet survey among Canadian adults found a mean daily intake of 13.8 µg/day (range of 7-34 µg/day) or 0.21 µg/kg bw/day (Dabeka et al., 1987). In comparison, reported mean dietary cadmium intakes in the U.S. include 28 µg/day in 1982 (Gartrell et al., 1986) and 15-20 µg/day (U.S. EPA, 1981). The food exposure for adults and children is assumed to be the same, although realistically it may be expected that this intake will vary with individual dietary consumption habits.

8.3.1.5 Smoking

Smoking can increase cadmium exposure in adults. It is estimated that individuals smoking one pack of cigarettes a day have cadmium blood and body burdens approximately twice that of non-smokers (Sharma et al., 1983; Lewis et al., 1972). This is estimated from an intake of 1-3 µg/day. The total daily intake estimates presented do not take this particular exposure into account when estimating the likely environmental exposure.

8.4 RISK CHARACTERIZATION

The modelled exposure from individual pathways and resultant total daily intakes for adults and young children receptors are summarized in Table 24. It is expected that exposure will be predominantly through normal dietary intake, accounting for almost all daily exposure in both children and adults. The relative contribution from the soil/dust pathway is preduicted to be less than 1% of average daily exposures.

The discrete soil intakes for children and adults are well below the reported Reference Dose values for cadmium in food estimated by EPA on the basis of nephrotoxicity (see Figure 9). They are also below the acceptable levels based on the WHO provisional tolerable weekly intake for cadmium. Total estimated intakes for adults and children also fall within acceptable limits.

It is therefore concluded that exposure to camium in soil at the reported levels will represent only a trivial fraction of total exposure and that such exposure would not be anticipated to pose a hazard to persons in these areas.

TABLE 24 ESTIMATED DAILY INTAKE OF CADMIUM: INTEGRATION OF EXPOSURE PATHWAYS

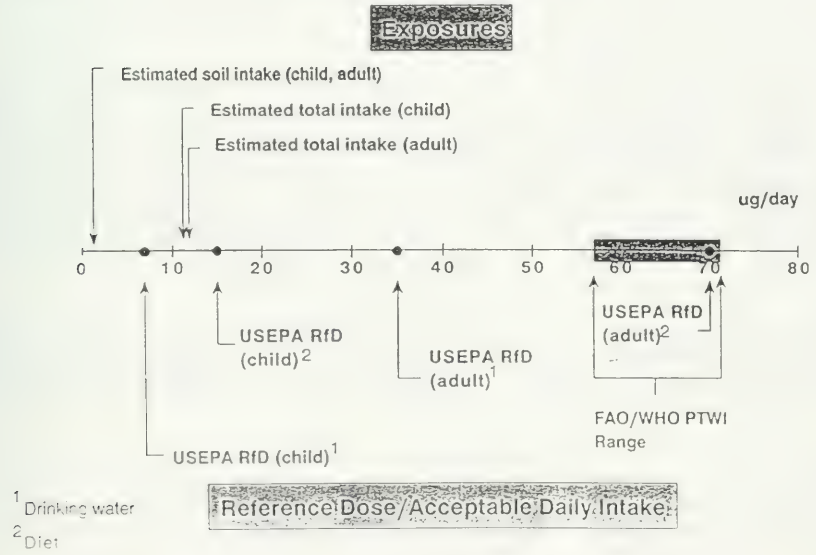
Substrate or Medium	Child (1-6 years old)	Adult
SOIL/DUSTS Average Soil Concentration	0.5 µg/g	0.5 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake from Soil	0.04 µg/day	0.01 µg/day
AIR Average Concentration in Air (Urban Ontario)	0.003 µg/m^3	0.003 µg/m^3
Volume Inhaled	5 m^3/day	22 m^3/day
Estimated Inhaled Intake	0.02 µg/day	0.07 µg/day
DRINKING WATER Assumed Tap Water Concentration	0.3 µg/L	0.3 µg/L
Daily Water Consumption	0.6 L	1.5 L
Estimated Intake from Drinking Water	0.2 µg/day	0.5 µg/day
FOOD Total Intake	13.8 µg/day	13.8 µg/day
ESTIMATED TOTAL DAILY INTAKE	13.9 µg/day	14.4 µg/day

TABLE 25 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate or Medium	Percentage of Total	
	Child (1-6 years old)	Adult
Soil/Dust	0.3	0.1
Air	0.1	0.5
Drinking Water	1	3.5
Food	99	96
TOTAL	100%	100%

FIGURE 9:

RISK CHARACTERIZATION
(EXPOSURE AND RfD/ADI'S FOR Cd)



9 COBALT

9.1 HAZARD IDENTIFICATION

9.1.1 Absorption and Metabolism

Cobalt exists in nature as the metal and in two valence states - CoII and CoIII, which forms numerous organic and inorganic salts. Cobalt is an essential nutrient functioning as a cofactor for several enzymes, and it is required for the synthesis of vitamin B_{12} (MOL, 1988).

Man absorbs about 25% of ingested soluble cobalt salts, with great individual variation, but more than 75% of the amount in food (ICRP, 1984; MOL, 1988). Cobalt and its salts are readily absorbed in the gastro-intestinal tract but the degree is dependent on dose where the amount absorbed decreases with increasing dose. Absorption also seems to be dependent on diet (ICRP, 1984; MOL, 1988; Stokinger, 1981b). In animals where large doses of cobalt are given orally approximately 80% is excreted in the feces and the remainder in the urine (Taylor and Marks, 1978 as cited in Domingo, 1989; MOL, 1988). In humans, the absorbed cobalt is excreted predominately through the urine, with about 10% in the feces and some in sweat (ICRP, 1984; MOL, 1988). The initial excretion is rapid, but some may be retained for several months.

9.1.2 Toxicology

Cobalt salts, like copper salts, in sufficiently large doses can cause gastrointestinal tract irritation. Acute exposures in patients receiving cobalt for treatment of anemia have shown symptoms of hypothyroidism, nausea, tinnitus and neurogenic deafness. Cobalt may also elicit other neurotoxic effects and cardiotoxic effects. Polycythemia (elevated red blood cell level) is the characteristic response of most mammals, including humans, to ingestion of excessive amounts of cobalt.

Small epidemics of severe cardiomyopathy have been observed resulting from heavy consumption of beer to which cobalt compounds had been added. Doses may have been as high as 10 mg per day (Friberg, 1986).

Inhalation of cobalt and compounds in the occupational settings has been associated with various effects. Occupational exposures to dusts containing cobalt mixed with other materials can cause a severe type of pneumoconiosis as well as obstructive lung disease at concentrations of >60 µg Co/m^3. Allergic dermatitis has also been reported in workers exposed to cobalt-containing materials (MOL, 1988; Stokinger, 1981b).

Cobalt has not been shown to cause significant teratogenic or reproductive effects in humans, although some <u>in vitro</u> studies have

been positive. Oral administration of cobalt did not produce teratogenicity or significant fetotoxicity in the rat at daily doses as high as 100 mg $CoCl_2$/kg (Domingo, 1989).

There is very little information available on the mutagenicity of cobalt, but the available data do not suggest that it has strong mutagenic properties.

Single or repeated injections of cobalt powder or cobalt salts have induced malignant tumours at the site of injection in rats but not in mice. There was no increase in the incidence of lung tumours in hamsters exposed to cobalt oxide dust as compared to controls. To date induction of cancer in experimental animals has not been possible except by injection. Epidemiological evidence for the carcinogenicity of inhaled cobalt compounds among industrial workers is conflicting because the exposure has been to a mixture of dusts. Therefore, it cannot be concluded that there is a correlation between occupational exposure to cobalt and cancer.

According to a review by Domingo (1989), most authors conclude that cobalt poses no recognized health hazard at environmental concentrations to nonocuupationally exposed individuals.

9.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

No drinking water objectives have been set for Ontario. It has been reported that young rats exposed to $CoCl_2$ in drinking water at a concentration of 500 ppm for three months showed various adverse effects including hypertrophy of the spleen, lungs and heart, and decrease in the values of the nutritional parameters. Studies in rats involving chronic drinking water exposure at a level of 2 mg Co/L have been shown to effect the erythropoietic system, cause immunosuppression and inhibit reflex learning (Carson et al., as cited in Mol, 1988). These effects were not seen at 200 µg/liter doses.

An ambient air guideline of 100 ng/m^3 has been proposed (MOL, 1988) based on a study where a LOEL of 100 µg/m^3 was reported miniature swine. This guideline incorporates an uncertainty factor of 10 because a LOEL instead of NOEL was used to account for interspecies variation and variation with in the human population.

9.3 HUMAN EXPOSURE ASSESSMENT

Cobalt exists probably only as CoII in both soil and water, and CoIII is unstable in aqueous media (Stokinger, 1981b).

9.3.1 Estimated Intake from Individual Sources

9.3.1.1 Soil and Dust

The sampling of soil in Port Hope in 1987 indicates that the horizontal distribution of cobalt is rather uniform, with a few random elevated concentrations. Of the 41 samples, only one was >25 µg/g. From the 1987 data (0-5 cm) the following values were determined:

- average concentration 8 µg/g (n=41)
- minimum concentration 0.1 µg/g
- maximum concentration 38 µg/g

Based on these concentration, the estimated cobalt intakes from soil/dust were calculated and are shown in Table 26.

TABLE 26 ESTIMATED INTAKES OF COBALT FROM SOIL/DUST INGESTION

	Child (1-6 years old)	Adult
Soil Concentrations Average Range	8 µg/g 0.1 µg/g-38 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	0.6 µg/g	0.2 µg/g
Minimum Intake	0.008 µg/day	0.002 µg/day
Maximum Intake	3.0 µg/day	0.8 µg/day

9.3.1.2 Air

The concentration at Port Hope is not known. It has been estimated that the intake is <0.1 µg/day (ICRP, 1984).

9.3.1.3 Drinking Water

The mean concentration of cobalt in Lake Ontario is around 0.05 µg/L (Rossman and Barres, 1988). Data from the Oshawa treatment plant indicate that cobalt levels are almost all below the detection limit of 1 µg/L. Therefore, the value of 0.05 µg/L was assumed for exposure analysis. Estimated intakes for adults and children are outlined in Table 27.

9.3.1.4 Food

There is only meagre information available on the daily intake. The average intake is around 300 µg/day, and the maximum close to 600 µg/day (ICRP, 1984; MOL, 1988; Stokinger, 1981b). Dietary intakes as high as 1800 µg/g have been suggested.

TABLE 27 ESTIMATED DAILY INTAKE OF COBALT: INTEGRATION OF EXPOSURE PATHWAYS

Substrate or Medium	Child (1-6 years old)	Adult
SOIL/DUST		
Average Soil Concentration	8 µg/g	8 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake	0.6 µg/day	0.2 µg/day
AIR		
Average Concentration	N/A	N/A
Volume Inhaled	5 m^3/day	22 m^3/day
Estimated Inhaled Intake	<0.1 µg/day	<0.1 µg/day
DRINKING WATER		
Assumed Tap Water Concentration	0.05 µg/L	0.05 µg/L
Daily Water Consumption	0.6 L/day	1.5 L/day
Estimated Intake	0.03 µg/day	0.08 µg/day
FOOD		
Estimated Intake	300 µg/day	300 µg/day
ESTIMATED TOTAL DAILY INTAKE	301 µg/day	300 µg/day

TABLE 28 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate or Medium	Percentage of Total	
	Child (1-6 years old)	Adult
Soil/Dust	0.2	0.07
Air	0.03	0.03
Drinking Water	0.01	0.03
Food	99.7	99.8
TOTAL	100%	100%

9.4 RISK CHARACTERIZATION

The toxicological information regarding the chronic effects of cobalt exposure via ingestion is limited. No specific reference doses or tolerable intake values for chronic human exposure were located.

It is assessed that cobalt exposure from soil is negligible when compared to total exposure. Food accounts for >99% and soil accounts for only a fraction of a percent of total exposure (see Tables 27 and 28). The exposure from food, taken to 300 µg/day for both adults and children, is highly variable and the reported data range from 300 µg/day to 1800 µg/day (Calabrese et al., 1985 as cited by Domingo); hence, the soil intake estimate, as a percentage of total exposure, is likely to even smaller.

Estimated exposures from air and water are also negligible with both estimated to be 0.03% of total exposure.

Excess exposures are not expected at these enviromental concentrations. On the basis of this exposure assessment, no adverse health effects as a consequence of cobalt exposure from soils in Port Hope is predicted.

10 SELENIUM

10.1 HAZARD IDENTIFICATION

10.1.1 Absorption and Metabolism

Selenium occurs in nature as -2 (selenides), 0, 2, 4 (selenites) and 6 (selenates). The last two are the most prevalent. Since most selenium salts are water soluble, they and the organic forms are taken up rapidly from the gastro-intestinal tract (40-80% absorption) and the lungs (approximately 95% absorption). Slightly soluble forms can also be taken up after deposition in the lungs. It is methylated in the liver. The dimethylated form is eliminated in the breath whereas the trimethylated is excreted in the urine. These are the major elimination pathways at high or toxic doses. At trace doses and deficiency states, selenium is eliminated via the feces. Some is also eliminated in sweat. Elimination has a rapid phase ($t_{1/2}$ = 1-3 days) and two slower ones ($t_{1/2}$ = 8-20 days and 65-116 days).

10.1.2 Toxicology

Selenium is believed to be an essential nutrient in animals and, most likely, humans. Man needs 50-70 µg/day to replace losses. Chronic toxicity is affected by the nutritional state of the animal and the interactions with other trace elements or nutrients. Chronic symptoms in animals, which appear at a dose rate >0.5 mg/kg/day, include reduced body weights and growth rate and death. Changes to some organs and in enzyme activity have been reported. Humans consuming more than about 1.5 mg/kg (about 20 µg/kg/day) could have the following symptoms: loss of hair and nails, dental caries, skin lesions and depigmentation, and effects on the nervous and gastro-intestinal systems. Studies of populations in China have shown that an intake of around 11 µg/day leads to symptoms of selenium deficiency; that an intake of around 5,000 µg/day leads to symptoms of selenosis and that an intake of 750 µg/day did not result in any symptoms.

Studies of interactions between selenium and other trace metals have shown that, in the case of some metals, such as methylmercury, selenium can protect against metal toxicity. There have been, however, few investigations on humans. It has been suggested that selenium has a protective effect against cancers caused by other trace metals.

Mammals exposed to doses which are near toxic levels for adults are found to have reduced fertility and fetal development. Reports of teratogenic effects in mammals are quite rare for mammals but more common for birds. It has been suggested that selenium may also be a teratogen in humans (Robertson, 1970).

The majority of short-term bacterial assays for gene mutations are

negative. Chromosome aberrations have been detected in mammalian cells in vitro and in whole animals. Tests to detect damage to DNA of bacteria and cultured mammalian cells have generally been positive. Selenites are more active than selenates.

IARC has determined that the available data are insufficient to allow an evaluation of carcinogenicity in animals. The older positive tests are of poor quality and the newer tests have not found selenium to be carcinogenic. Only selenium sulfide administered by gavage and dermally has been found to cause cancer in male mice. The available epidemiological data provide no suggestion that selenium is carcinogenic in man.

10.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

The U.S Food and Nutrition Board has recommended a safe and adequate daily dietary intake range of 0.05 to 0.2 mg per person per day for adults and correspondingly lower ranges for infants and children (Robinson et al, 1979). The dietary requirement below which adverse health effects may occur has been estimated at 0.02 to 0.12 mg/day (NAS, 1976; Stewart at al, 1978).

The current maximum acceptable concentration for selenium in drinking water has been established at 0.01 mg/L on the basis of health considerations (HWC, 1986). In the derivation of this guideline the minimum toxic intake level for adults of 0.5 to 0.7 mg/day is considered (Sakurai and Tsuchiya, 1975; Allegrini et al, 1988). This would be equivalent to 0.1 to 0.15 mg/day for a 15 kg child, adjusting only for weight.

10.3 HUMAN EXPOSURE ASSESSMENT

Selenium exists in inorganic and organic forms in soil. In acid or neutral soils as are found in humid areas, SeIV combines with Fe III to form compounds of very low solubility. In alkaline soils under semi-arid conditions, it exists as water-soluble selenates, which do not form stable complexes or insoluble salts. Less than 10% of the selenium in soils is water soluble.

Almost all selenium is in soluble form; a small percentage particulate.

Selenium is bound to fly ash and other suspended particles in urban air.

10.3.1 Estimated Intake from Individual Sources

10.3.1.1 Soil and Dusts

The analysis of the soils sampled in Port Hope around the Eldorado Resources Ltd. plant in 1987 indicates that the distribution of total selenium in the 0-5 cm layer of the soil is quite uniform

with distance from the plant. There are a few random elevated concentrations. Of the 41 surface samples, only 6 were >0.5 µg/g (air dried). From the 1987 data (0-5 cm) the following values were determined:

- average concentration 0.31 µg/g (n=41)
- minimum concentration 0.02 µg/g
- maximum concentration 1.0 µg/g

Based on these concentrations, the estimated selenium intakes from soil/dust were calculated and are shown in Table 29.

TABLE 29 ESTIMATED INTAKES OF SELENIUM FROM SOIL/DUST INGESTION

	Child (1-6 years old)	Adult
Soil Concentrations Average Range	0.31 µg/g 0.02 µg/g-1.0 µg/g	
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Average Intake	0.025 µg/day	0.006 µg/day
Minimum Intake	0.002 µg/day	0.0004 µg/day
Maximum Intake	0.08 µg/day	0.02 µg/day

10.3.1.2 Air

The ambient air values for selenium in total suspended particulates in Ontario is not known. It is estimated that ambient air in urban areas contains 0.1-10 ng/m^3. The midpoint of approximately 5 ng/m^3 is assumed.

10.3.1.3 Drinking Water

No treated drinking water concentrations for selenium were located. The median concentration of selenium in Lake Ontario is 1 µg/L. This value was therefore assumed but may overestimate treated water levels.

10.3.1.4 Food

There is incomplete information on the chemical form and absorption of selenium in foods, but apparently 40-80% is absorbed.

The average dietary intake (1973) in the USA is estimated to be 150 µg/day. A 1975 Canadian study indicated an average intake by adults

of 150 µg/day ranging 100 to 225 µg/day. The major sources are grain and cereal (about 60%) and meat, fish and poultry (approx. 40%). Dairy products are a minor source and most other food groups contribute nothing. Results from U.S. total diet studies provide averages of adult intake of 139 µg/day and intakes for toddlers and infants of 54 µg/day, respectively (Gartrell et al, 1986 a,b). The values of 150 µg/day and 54 µg/day are assumed for adults and young children.

TABLE 30 ESTIMATED DAILY INTAKE OF SELENIUM: INTEGRATION OF EXPOSURE PATHWAYS

Substrate or Medium	Child (1-6 years old)	Adult
SOIL/DUST		
Average Soil Concentration	0.31 µg/g	0.31 µg/g
Soil/Dust Ingested	80 mg/day	20 mg/day
Estimated Intake from Soil	0.025 µg/day	0.006 µg/day
AIR		
Average Air Concentration (Urban)	0.5 ng/m^3	0.5 ng/m^3
Volume Inhaled	5 m^3/day	22 m^3/day
Estimated Intake	0.025 µg/day	0.1 µg/day
DRINKING WATER		
Assumed Tap Water Concentration	1 µg/L	1 µg/L
Daily Water Consumption	.6 L/day	1.5 L/day
Estimated Intake	.6 µg/day	1.5 µg/day
FOOD		
Total Intake	22 µg/day	54 µg/day
ESTIMATED TOTAL DAILY INTAKE	22.6 µg/day	55.6 µg/day

10.4 RISK CHARACTERIZATION

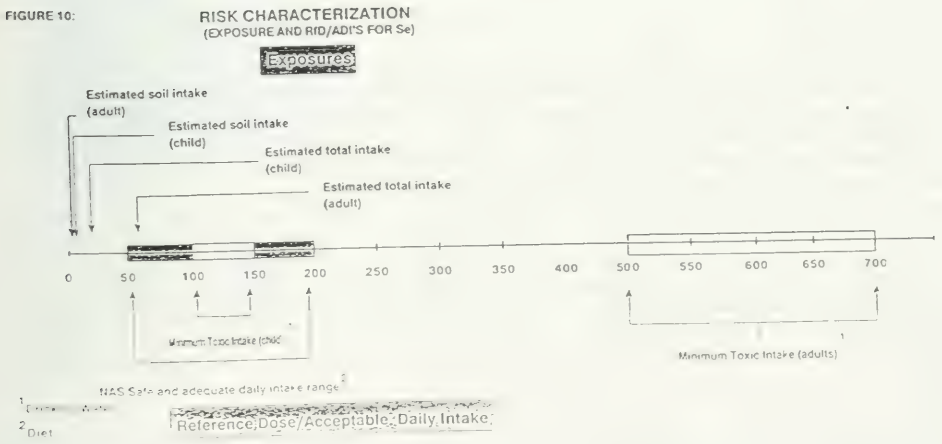
Intake of selenium from soil is estimated to be only a trivial fraction of total exposure as shown in Table 30. The total intakes of selenium from all sources (22.6 µg/day for a child and 55.6 µg/day for an adult) are also well below the minimum toxic intake levels of 500-700 µg/day for adults and 100-150 µg/day for a child.

No adverse health consequences would be predicted to result from intake of selenium in soils at the levels reported in Port Hope on the basis of this exposure assessment.

TABLE 31 RELATIVE CONTRIBUTIONS OF EACH EXPOSURE PATHWAY

Substrate of Medium	Percentage of Total	
	Child (1-6 years old)	Adult
Soil/Dust	0.1	0
Air	0.1	0.3
Drinking Water	2.8	2.7
Food	97	97
TOTAL	100%	100%

FIGURE 10:



11 ZINC

Rigorous assessment of potential exposures to this metal was not undertaken because of its known low toxicity.

Zinc is a nutritionally essential trace element for both animals and man, and is estimated to be found at levels of 2-3 g in the normal adult man and is found in all body tissues. It has been associated with over 200 zinc containing metalloenzymes (Falchuk and Vallee, 1985) and it therefore functions in the metabolism of carbohydrates, proteins and nucleic acids. Most health consequences related to zinc are a result of deficiencies in the body. Prasad (1983) has described the clinical manifestations of chronic zinc deficiency in third world children.

Excessive exposures to zinc are uncommon. Homeostatic mechanisms in the body related to absorption efficiently modulate zinc levels and it therefore does not accumulate in the body with continued exposure (NRCC, 1981, Prasad, 1983). Zinc overload is therefore extremely unlikely.

Zinc and zinc compounds exhibit relatively low toxicity by ingestion (Cassaret and Doull, 1986; NAS , 1977). Reports of zinc toxicity are primarily anecdotal. Swallowing large amounts of zinc powders or dust may cause inflammation of the stomach (gastritis) and vomiting. Large intakes of zinc have also been demonstrated to impair immune response in individuals receiving 150 mg twice daily for six weeks (Chandra, 1984). High oral intakes may also affect copper metabolism (NHW, 1990). Occupational exposure involving inhalation of zinc oxide fumes has been reported to induce metal fume fever in workers.

There is no evidence to suggest that zinc is carcinogenic.

In assessing the levels of zinc measured in soil in Port Hope, it is considered that, with respect to human health in the general population, that any concern would not be with exposure and toxicity, but rather marginal or deficient intakes. To look at the question somewhat more quantitatively, one can look at the highest measured zinc concentration reported (4330 µg/g at site # 49, 5-10 cm in 1987). Assuming an 80 mg soil ingestion rate yields a calculated possible intake of 0.35 mg/day. This value is several fold lower than recently published nutrition recommendations for zinc (see Table 32) prepared by the Scientific Review Committee of Health and Welfare Canada. These values range from 2 to 12 mg/day depending on age and sex. Such an intake is also only a small fraction of estimated intakes of zinc provided in the Canadian diet.

The nutritional essentially, homeostatic considerations, low toxicity and comparison to dietary levels and currently recommended nutrient intakes for Canadians together indicate that the reported

zinc levels measured in Port Hope would not represent an adverse health impact for persons exposed to these soils.

TABLE 32 RECOMMENDED NUTRIENT INTAKE BASED ON AGE

AGE	IRON INTAKE (MG/DAY)	ZINC INTAKE (MG/DAY)
0-4 mo	0.3	2
5-12 mo	7	3
1-3 yr	6	4
4-6 yrs	8	5
7-9 yrs	8	7
10-12 yrs	8	9
13-15 yrs		
males	10	12
females	13	9
16-18 yrs		
males	10	12
females	12	9
19-49 yrs		
males	9	12
females	13	9
50- yrs		
males	9	12
females	8	9
Pregnancy		
1st Trimester	0	6
2nd Trimester	5	6
3rd Trimester	10	6
Lactation	0	6

Part II RADIONUCLIDES ASSESSMENT

Table 12 of the Phytotoxicology report gives the activities of various radionuclides in the surface soil of 23 sites.

The activities of ^{228}Thorium (^{228}Th) and ^{228}Radium (^{228}Ra) did not exceed the detection limit of 20 mBq/g of soil. Their concentrations in soil could thus not be determined but were less than 20 mBq/g. It should be noted that the half-lives of these two isotopes are 1.9 years and 6.7 years respectively, so that a contamination introduced into the soil will disappear relatively rapidly.

The results of the measurements of ^{40}Potassium (^{40}K) listed in Table 12 (Phytotoxicology Report) could be described, statistically, either by a normal distribution, with a mean of 520 and standard deviation of 134, or by a lognormal distribution, with a geometric mean of 6.21 (corresponding to 500 mBq/g) and a geometric standard deviation of 0.34. The mean concentrations were thus less than the area farmland mean of 630 mBq/g. ^{40}K is a naturally occurring radionuclide with a half-life of 1.3 billion years. Potassium occurs in a wide range of concentrations in rocks and soils. The potassium content of soils of arable lands is controlled by the use of fertilizers, since radioactive potassium is present to the extent of 30 Bq per gram of stable potassium. It thus appears that the concentration of ^{40}K in the soils of Port Hope is not elevated beyond the concentration found in area farmland.

The measurements of ^{226}Radium (^{226}Ra) and ^{210}Lead (^{210}Pb) listed in Table 12 (Phytotoxicology Report) exceed the background levels. The table gives the mean value of ^{226}Ra to be 210 mBq/g. The measurement values are, however, skewed, and the statistical distribution is better described by a lognormal distribution than by a normal distribution. The geometric mean for radium is 110 mBq/g, higher than the area farmland mean and the background quoted from the literature. The geometric mean for ^{210}Pb is 158 mBq/g, again higher than the area farmland mean and the background literature. It may thus be concluded that the soils sampled are contaminated with ^{226}Ra and ^{210}Pb.

1 ^{226}Ra and ^{210}Pb EXPOSURE

^{226}Ra is an isotope of radium with a half-life of 1,600 years. It is absorbed from the intestinal tract and stored in bone, where it may present a hazard by induction of bone tumours. ^{210}Pb is an isotope of lead with a half-life of 22 years. It is absorbed from the intestinal tract by induction of bone tumours.

The main routes for human exposure to the radionuclides in these contaminated soils are consumption of garden produce and ingestion of soil, wether directly or by inhalation of dust.

1.1 Consumption of Garden Produce

In 1983, the Radiation Protection Bureau of Health and Welfare Canada (Tracy et al., 1983) analyzed the risk to humans from the consumption of garden produce grown in contaminated soils in Port Hope. The gardens they considered were generally more contaminated with radium than the soils listed in Table 12 (Phytotoxicolgy Report), with radium concentrations ranging from 370 to 30,000 mBq/g. The authors combined information obtained from dietary questionnaires with measurements of radium concentration in the garden produce to estimate radiation doses to the bones of the consumers. The doses estimated from this calculation were generally well less than 1% of the population dose limit recommended by the International Commission on Radiological Protection (ICRP, 1990).

The authors did not present a similar calculation for ^{210}Pb, but a risk estimate can be inferred from the data presented. Lead is taken up less readily than radium by garden produce, the radioactivity in plant tissues due to lead being only about one-sixth of the activity due to radium. The ICRP has calculated that, because of its somewhat higher toxicity, the allowable annual intake for ^{210}Pb should by only 2/7 of the allowed intake for radium (ICRP, 1980). Since the measured activity of ^{210}Pb in garden produce is only about one-sixth of the activty of radium, one may infer that the dose from ^{210}Pb due to ingestion of garden produce will be similar in magnitude to the dose from radium in the produce, i.e. generally less than 1% of the population dose limit.

Adding together the doses from lead and radium in produce, one may infer that the combined annual dose from these radionuclide is probably substantially less than 1-2% of the population dose limit.

1.2 Soil Ingestion

Soil may be ingested directly, from contaminated hands or objects, or inhaled dust may be swallowed. This route is primarily a concern with respect to the exposure of children.

A model in which children ingest 80 mg of soil per day and adults ingest 20 mg per day is assumed. If we assume that the soil contains 200 mBq of each of ^{226}Ra and ^{210}Pb per gram of soil, then according to this model, a child might ingest about 20 mBq of each of these radionuclides per day. This might result in an annual ingestion of 7 Bq of each of these radionuclides per year. The dosimetry and risk estimation for these radiochemicals in children are uncertain. One crude comparison might be to compare these uptake figures with the annual limits of intake (ALI) for 70 kg adult workers. The ALI's are 20,000 Bq per year for ^{210}Pb and 70000 Bq per year for ^{226}Ra. The assumed intakes of 7 Bq per year thus correspond to 0.04% of the ALI for ^{210}Pb and 0.01% of the ALI for ^{226}Ra.

CONCLUSIONS

Elevated levels of ^{226}Ra and ^{210}Pb have been found in soil samples from Port Hope. If ingested, these radioisotopes are stored in bone and present the potential risk of induction of bone cancer. The major ingestion pathways are the consumption of garden produce and ingestion of contaminated soil. It is concluded that under reasonable assumptions about the quantities of produce and soil ingested, the annual radiation dose attributable to these radionuclides will be less than several percent of the population limits recommended by the International Commission on Radiological Protection and the Atomic Energy Control Board.

Elevated levels of ^{238}uranium (^{238}U) were also measured in the survey as discussed above under uranium toxicity (pg. 47). It is similarly calculated that the annual intake attributable to ^{238}U activity is substantially less than 1% of the recommended intake limits.

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APPENDIX I - Soil Ingestion Rates

Faced with relatively little research evidence, it has been traditional to assume or crudely estimate a value for the amount of soil/dust ingested by young children. Typical average ingestion rates of this type range from 50 to 250 mg/day for children 2-3 years old (Hawley, 1985). A number of documents have used an average estimate of 100 mg of dirt ingested daily by children to represent a probable value for a "typical" child. Children suffering from pica may ingest more than 1 g/day (NAS, 1980).

Biokinetic modelling studies of lead intakes have suggested that 50 to 60 mg/day for ingestion of soil and dust is a more accurate estimate (Huffnage, 1988).

Recent progress in examining this question has involved the utilization of epidemiological mass balance studies in young children (Clausing et al, 1987, Binder et al., 1986; Calabrese et al., 1989, 1990; Davis et al., 1990; van Wijnen et al., 1990. The most comprehensive of these are the Calabrese et al., 1989 and Davis et al., 1990 studies. These studies essentially measure the fecal excretion of relatively non-absorbed tracer elements found in soil (including aluminum, silicon, zirconium and titanium). These measurements are then utilized in mass-balance equation to derive an estimate of soil intake. Various tracers yield a range of estimates both within and between studies.

The principle finding of the 1989 Calaberse et al. study were that the median soil intake value based on all eight tracers ranged form 9 to 96 mg/day, with seven of the eight tracers having median values less than 50 mg/day. The three most relaible tracers revealed a range of median values of from 9 to 40 mg/day of soil ingested. The Davis et al. work indicated median daily estimates of 25.3 mg/day based on aluminum, 59.4 mg/day based on silicon and 81.3 mg/day based on titianium. Mean values were higher at 38.9, 82.4 and 245.5 mg/day for Al, Si, and Ti respectively.

Estimates based on titanium are typically highly variable and may be as much as an order of magnitude larger than those with other tracers. Calabrese has concluded that Ti estimated values in studies which lack food ingestion data may significantly overestimate soil ingestion (4-6X for Calabrese data if mass-balance approach not used). The authors conclude that the discepancy is largely based on high levels of Ti in food and that the three most reliable tracers are Al, Si, and Y based on validation in adult volunteers. It has been suggested that the titanium-based estimates be treated as outlyiny and given less weight (U.S. EPA, 1989b).

From this information a value of 80 mg/day soil ingested for children is selected for the exposure estimates for this pathway.

There is no empirical data regarding soil ingestion rates for adults. A value of 20 mg/day is used, as adults will not have the same degree of hand-mouth activity. These values are in keeping with the recommended Canadian reference values for dirt, dust and soil intake (HWC, 1988).

APPENDIX II

MODELLED INTAKES OF INORGANICS THROUGH HOMEGROWN GARDEN PRODUCE

Modelling of this exposure route requires two sets of assumptions:

1) bioavailability and uptake to plants from soil for each contaminant and

2) estimate of the consumption rates for homegrown produce. The intake is given by the algorithm

$$\text{Intake} = C_{food} \times IR$$

where c is the contaminant concentration in food (fresh weight, mg/kg) and IR is ingestion rate in kg/day.

Modelled produce concentration is calculated as a function of soil concentration (mean measurement) and bioavailability. This is compared against the reported range of critical tissue concentrations for each contaminant (this critical level of an element is defined as the lowest concentration at which it has toxic effects and yield is reduced). The concentration is considered the limiting concentration for exposure estimates described in the attached table. The assumptions regarding consumption of vegetables are as follows. The amounts and types of produce that people might consume from a backyard garden are influenced by the size of the garden, the yields of the crop grown, and the preferences of the individuals. The actual dose received will also be lowered by the reductions of concentrations in produce that result when produce is washed, peeled and boiled.

According to the produce module of the AERIS (Aid for Evaluating the Redevelopment of Industrial Sites) Model for exposure, the yields of various types of crops generally range from 0.3 to 2.6 kg/m^2. For a mixture of crops that might be found in a backyard garden, a yield of 1.4 kg/m^2 has been recommended, although it is possible to achieve higher yields with special techniques or extraordinary efforts. The model assumes that a garden area is 30 m^2 with a yield of 1.4 kg/m^2; this gives a total of 42 kg, which represents 13% of the vegetables and fruits that one adult and one child would consume in one year. If one assumes a family size of four, then approximately 7% of fruits and vegetables would be garden-grown.

Nutrition Canada Survey (1972) data for Ontario indicate average daily consumption of fruits, fruit products and vegetables combined as 372 g/day (26% of total diet) for young children (one to four years) and 489 g/day (32% of total diet) for adult males, which convert to 136 kg/year and 178.5 kg/year respectively. Thus consumption of homegrown produce would be 26 g/day for children (1.8% of diet) and 34 g/day for adults (2.2% of diet).

TABLE II-1 MODELLED INTAKES THROUGH BACKYARD GARDEN PRODUCE CONSUMPTION

	Mean Soil Concentration (1986-1987)	Bioavailability Factor	Modelled/ Assumed Produce Concentration (µg/g, fresh weight)	Critical tissue Concentrations[3] (µg/g, fresh weight)	Intake from Produce, µg/day (absorbed intake)	Percentage of Estimated Average Daily Intake (%)
Arsenic child adult	20	4×10^{-3} [1]	0.08	0.1-1.8	2.1 (1.0) 2.8 (1.3)	14 15
Antimony child adult	53.5	-	0.2 [4]	0.1-0.2	2.1 (0.21) 2.8 (0.28)	ND ND
Chromium child adult	33	1×10^{-3} [2]	0.03	0.1-0.18	0.8 1.0	<1% <1%
Lead child adult	140	5×10^{-4} [3]	0.27	-	7.0 (7.0) 9.2 (9.2)	13-23 12
Nickel child adult	14	5×10^{-2} [1]	0.7	1.0-8.3	18.2 23.8	5 7
Copper child adult	38	6	1.0	0.5-3.5	26 34	
Uranium child adult	32	7.5×10^{-5} [4]	0.0025	-	.062 .085	13 25

[1]CARB Report
[2]Sheeham et al. (1991)
[3]MOE. Phytotoxicity section (unpublished data). Based on regression analysis.
[4]Tracey et al, 1983
[5]MacNiccoll, 1985
ND = not determinable

APPENDIX III

DERMAL ABSORPTION OF INORGANIC IONS

Human skin is made up of three layers: the stratum corneum (10μ), viable epidermis (100μ) and the papillary layer of the dermis (100-200μ). The permeability of the capillary walls to other than macromolecules is sufficiently great that diffusing molecules are readily absorbed into the capillary circulation. Hair follicles and sweat glands pierce this layer. However, their contribution to absorption of molecules and ions is probably negligible, except in the initial phase of absorption.

The stratum corneum is the main barrier to diffusion across the skin. This has been demonstrated experimentally. It is formed from dried, compact keratin-containing cells that are converted from aqueous epidermal cells. It is always partially hydrated and varies in thickness on the body.

The skin barrier can be regarded as a composite membrane, pierced over a small portion of its area by shunts of different but lower diffusivities. For a simple membrane, the steady-state flux of solute, J_s is given by

$$J_s = K_m D \blacktriangle C_s / \delta$$

where K_m is the solvent-membrane distribution coefficient, $\blacktriangle C_s$ is the concentration difference of solute across the membrane, D is the membrane diffusion coefficient for the solute and δ is the membrane thickness. The permeability coefficient, k_p, is defined as $K_m D/\delta$.

The permeability constant for water at 25°C is approximately 0.5×10^{-3} cm/hr, corresponding to a flux of 0.2 mg/cm^2/hr. The flux is approximately the same whether liquid water or saturated water vapour is applied in vitro.

Electrolytes in aqueous solution do not penetrate the skin as readily as water because:

- the stable hydration sphere around an ion makes it a much larger diffusing unit than a water molecule;
- the charge on the ion will interact with coions, counterions and fixed charges in the tissue.

Therefore, it is likely that, in the absence of a potential gradient, most ions will penetrate much more slowly than water, and no ion will penetrate faster. Measured permeability constants (k_p) are approximately same for many ions and are of the order of 10^{-6} cm/hr. Shunt diffusion through appendages may play a significant role.

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